Background Document B:

Chemicals Proposed for Regulation

Utah Chemical Agent Rule (UCAR)

May 1999

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FOREWORD

This document provides general background information on each of the chemical agents and associated chemicals that are proposed for regulation in some capacity in the proposed Utah Chemical Agent Rule (UCAR). For each chemical, relevant data and information are reviewed, concluding with a statement on how each chemical is proposed to be regulated.

The following chemicals are included in this document: GB, GD, GA, GF, VX, HD/H, L, HN1, HN3, HL, HQ, HT, DM, Q, T, Lewisite 2, Lewisite 3, EA2192, EMPA, TDG, MPA, IMPA, LO, Vx, CK, CG, and BZ.

HF, chloroform, and arsenic are also proposed as hazardous constituents for the chemical agent listings. HF is a breakdown product of the G agents. Chloroform is included because it is used as a solvent for several of the chemical agent identification sets (CAIS). Arsenic is a component of arsenical agents, including L and DM. These three chemicals are not included individually in the discussions below, however, because they are already listed as hazardous constituents under U.S. Environmental Protection Agency regulations (40 CFR 261, Appendix VIII), which the State of Utah Department of Solid and Hazardous Waste has adopted in R315-50-10.

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NOTATION

AChE	acetylcholinesterase
As	arsenic
BZ	3-quinuclidinyl benzilate
CAIS	chemical agent identification set
CAS	Chemical Abstracts Service
CAS Reg. No.	Chemical Abstracts Service Registry Number
CFR	Code of Federal Regulations
CG	phosgene, carbonyl chloride
ChE	cholinesterase
CK	cyanogen chloride
cm^2	square centimeter(s)
CN	chloroacetophenone
Ct	concentration $\times$ time
CVAA	2-chlorovinylarsonous acid
d	day(s)
DA	Department of the Army
°C	degree(s) Celsius
DM	adamsite, phenylarsazine chloride
DSHW	Division of Solid and Hazardous Waste (State of Utah)
DWEL	drinking water equivalent level
EA2192	S-(2-diisopropylaminoethyl) methylphosphonothioic acid
EMPA	ethyl methylphosphonic acid
EPA	U.S. Environmental Protection Agency
ERDEC	U.S. Army Edgewood Research Development and Engineering Center
F	waste code for waste listed in 40 CFR 261.31
g	gram(s)
GA	tabun, O-ethyl N,N-dimethyl phosphoramidocyanidate
GB	sarin, O-isopropyl methylphosphonofluoridate
GD	soman, O-pinacolyl methylphosphonofluoridate
GF	cyclohexyl methylphosphonofluoridate
μg	microgram(s)
H	sulfur mustard (Levinstein process, undistilled), bis(2-chloroethyl) sulfide

HCl	hydrochloric acid
HD/H	sulfur mustard or bis(2-chloroethyl) sulfide (unspecified purity)
HD	sulfur mustard (distilled), bis(2-chloroethyl) sulfide
HF	hydrogen fluoride, hydrofluoric acid
HL	mixture of mustard (HD) and lewisite
HN1	bis(2-chloroethyl)ethylamine
HN2	bis(2-chloroethyl)methylamine
HN3	tris(2-chloroethyl)amine
HOCl	hypochlorous acid
HQ	mixture of mustard (HD) and Q-mustard (Q)
HT	mixture of mustard (HD) and T-mustard (T)
HTH	high test hypochlorite
IMPA	isopropyl methylphosphonic acid
ICt_{50}	concentration × time that is incapacitation to 50% of an exposed population
K	waste code for waste listed in 40 CFR 261.32
K_a	acid dissociation constant
kg	kilogram(s)
K_H	Henry's Law constant
K_{oc}	organic carbon partition coefficient
K_{OW}	octanol/water partition coefficient
kPa	kilopascal(s)
L	liter(s)
L (or L1)	lewisite 1, 2-chlorovinyldichlorarsine
L2	lewisite 2, bis(2-chlorovinyl)chloroarsine
L3	lewisite 3, tris(2-chlorovinyl)arsine
LC_{50}	concentration lethal to 50% of an exposed population
LCt_{50}	concentration × time lethal to 50% of an exposed population
LD_1	dose lethal to 1% of an exposed population
LD_{50}	dose lethal to 50% of an exposed population
LD_{lo}	lowest dose causing a lethal effect
LO	lewisite oxide, 2-chlorovinylarsenous oxide
LOAEL	lowest observed adverse effects level
M	mole(s)
m^3	cubic meter(s)
mg	milligram(s)
min	minute(s)
mL	milliliter(s)
mol	mole(s)
MPA	methylphosphonic acid

MW	molecular weight
NAOH	sodium hydroxide
NOAEL	no observed adverse effect level
N.O.S.	not otherwise specified
P	waste code for waste listed in 40 CFR 261.33(e)
pK_a	negative logarithm of K_a (acid dissociation constant)
Q	Q-mustard, sesquimustard, 1,2-bis(2-chloroethylthio)ethane
RCRA	Resource Conservation and Recovery Act
s	second(s)
T	T-mustard, bis(2-chloroethylthioethyl) ether
T, t	temperature
U	waste code for waste listed in 40 CFR 261.33(f)
VX	O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothiolate
Vx	S-2(2-diethylamino)ethyl O-isobutyl methylphosphonothioate

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Background Document B: Chemicals Proposed for Regulation

1. GB (Sarin or O-Isopropyl Methylphosphonofluoridate) CAS Reg. No.[1] 107-44-8

CH_3-P(=O)(O-CH[CH_3]$_2$)F

A. Background

GB, an acutely toxic, relatively nonpersistent nerve agent, was developed and weaponized by the Germans during World War II but was never used. It was subsequently adopted by the U.S. armed forces, produced in quantity, tested in laboratories and in the open air, and loaded in munitions that were stockpiled (stored in 1-ton containers) but never employed in combat. GB was also included in some chemical agent identification sets (CAIS). However, these sets were all destroyed at Rocky Mountain Arsenal during the late 1970s and early 1980s, and no sets containing GB remain.

B. Physicochemical Properties

The following pressure-temperature relationship was reported for GB (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 7.48160 - 1{,}773.82/(227.9 + t[°C]).$$

GB evaporates at about the same rate as water (HQ/DA,DN,AF 1990). It undergoes fairly rapid loss from unconfined soils by evaporation, leaching, and hydrolysis (Small 1984).

The hydrolytic half-life of GB is highest (the rate slowest) in the pH range of 4-6, about 160 hours at pH 5 and 25°C, and decreases outside that range in either more alkaline or more acidic solutions (Clark 1989). The second order rate constant for hydroxyl-ion-catalyzed hydrolysis is (Demek et al. 1970):

$$\log k_2\ (M^{-1}min^{-1}) = 9.8507 - (1{,}985.4/t[K]).$$

This relationship gives a value of 1,543 $M^{-1}min^{-1}$ at 25°C. Hence, the estimated rate constant at pH 10 is 0.1543 min^{-1} and the half-life at that pH is 4 ½ minutes.

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[1] CAS Reg. No. = Chemical Abstracts Service Registry Number.

Small (1984) cited work by Puzderliski (1980) in an example of the rate of environmental disappearance of GB under relatively unfavorable conditions. GB in soil that was exposed to light rain at 0°C would disappear by a factor of 1,500 in 18 days. At 25°C, the time for this loss to occur was less than a day.

The strongly catalytic effect of aqueous hypochlorite ion on the hydrolysis of GB was studied by Epstein et al. (1956); they determined the second order rate constant to be 600 $M^{-1}min^{-1}$ at 25°C, with an acid dissociation constant (K_a) value of 4 x 10^{-8} for hypochlorous acid (HOCl). From these values, it can be estimated that the concentration of GB mixed with an excess of 3% commercial bleach at pH 8 would decrease over 1 million-fold in less than 5 seconds.

Environmentally relevant properties of GB are presented in Table 1. It may be concluded that, once released to the environment, GB would be expected to persist for relatively short periods of time. Moreover, GB can be detoxified with mild chemical treatment.

TABLE 1 Environmentally Relevant Properties of GB

Property	Data	Reference
Empirical formula	$C_4H_{10}FO_2P$	Not applicable
Molecular weight (MW), g/mol	140.1	Not applicable
Density, g/mL	1.0887/25°C	Samuel et al. 1983
Melting point, °C	-56.9	Samuel et al. 1983
Boiling point, °C	157.8	Samuel et al. 1983
Vapor pressure at 25°C, torr	2.94	Samuel et al. 1983
Log K_{ow}	0.15 (estimate)	Britton and Grant 1988
	0.299	Experimental value[a]
Aqueous solubility, g/L	Infinitely miscible	HQ/DA,DN,AF 1990
K_H, atm·m³/mol	4.0 x 10^{-7}/25°C	Estimate[b]
Log K_{oc}	1.8 (estimate)	Small 1984
	1.54	Calculated[c]

[a] See Appendix H of Background Document E.

[b] Based on estimated vapor pressure of 0.136 kPa for 0.1 mole fraction (ca. 3.34 M) of GB in water, K_H = 0.00134 atm/(3,340 mol/m³) from Figure 2 of Preston and Starrock (1983).

[c] Derived from Log K_{ow} of 0.299, as shown above, using equation from Lyman et al. (1982).

C. Toxicity

The toxicity of GB varies, depending on route of administration. The LCt_{50} (concentration x time that is lethal to 50% of an exposed population) for GB estimated for 70-kg humans breathing at the rate of 15 L/min is about 70 mg·min/m^3 (Edgewood Arsenal 1974). Assuming that GB is completely absorbed through the lungs, this value is equivalent to an oral LD_{50} (dose lethal to 50% of an exposed population) of about 15 µg/kg.

Table 2 shows the lethal effects of GB in experimental animals by different routes of exposure.

Like all nerve agents, GB exerts its effects through inhibition of the enzyme acetylcholinesterase (AChE), which is required for nerve and muscle function. Normally, AChE prevents accumulation of acetylcholine after its release as a chemical messenger in the nervous system. AChE inhibition adversely affects skeletal muscle, parasympathetic end organ, and central nervous system operation. Individuals poisoned by sufficient amounts of GB may show the following signs and symptoms soon after exposure (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990):

- Difficulty in breathing, tightness of chest;
- Dimness of vision and pinpointing of the eye pupils;

TABLE 2 Comparison of Some Toxicities of GB by Different Routes of Exposure in Different Species

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Mouse	113 µg/kg
Intravenous	LD_{50}	Rat	39 µg/kg
Inhalation	LD_{50}[a]	Mouse	5 mg/m^3/10 min
Subcutaneous	LD_{50}	Guinea pig	30 µg/kg
Subcutaneous	LD_{50}	Rat	103 µg/kg
Subcutaneous	LD_{50}	Rabbit	30 µg/kg
Intraperitoneal	LD_{50}	Rat	218 µg/kg
Intraperitoneal	LD_{50}	Mouse	420 µg/kg

[a] LC_{50} = concentration lethal to 50% of an exposed population.

Source: Sweet (1987).

- Drooling and excessive sweating;
- Nausea;
- Vomiting, cramps, and loss of bladder/bowel control;
- Twitching, jerking, and staggering;
- Headache, confusion, drowsiness, coma, and convulsion; and
- Death.

An important characteristic that distinguishes behavior of GB from other nerve agents is that cholinesterase (ChE) inhibited by GB can undergo spontaneous reactivation, whereas ChE inhibited by some of the other nerve agents rapidly "ages" to an inhibited form of the enzyme that is resistant to either spontaneous or therapeutic reactivation (Daniels 1990).

The number and severity of symptoms of GB poisoning depend on the quantity and route of entry of this nerve agent into the body, as well as the duration of exposure. When the eyes are exposed to the agent vapor, a prominent sign is pinpointing of the pupils. Dimness of vision results from the reduced amount of light entering the eyes. However, if exposure to the nerve agent is through the skin or by ingestion, the pupils may be normal or only slightly reduced in size. In this event, diagnosis must rely on other symptoms of nerve agent poisoning (HQ/DA,DN,AF 1990).

Respiratory exposure usually results in the onset of symptoms in 2 to 5 minutes; lethal doses kill in less than 15 minutes. Exposure through the eyes also produces a very rapid onset of symptoms (usually less than 2 to 3 minutes). Liquid agent in the eyes kills nearly as rapidly as respiratory exposure. Symptoms from skin absorption appear more slowly. Death from skin absorption may occur in 1 to 2 hours. Very small skin dosages sometimes cause local sweating and tremors (muscle twitching) but few other effects (HQ/DA,DN,AF 1990).

If a victim recovers from acute GB poisoning, the recovery will be complete unless anoxia and convulsions have gone unchecked so long that irreversible central nervous system damage has occurred (HQ/DA,DN,AF 1990).

Ninety-day subchronic toxicity test results for GB in male rats gave a lowest observed adverse effects level (LOAEL) of 0.075 mg/kg/d (5 days per week, adjusted to 0.054 mg/kg/d for 7 days per week) of agent administered by gavage. At the end of the first week of exposure, red blood cell cholinesterase was depressed 38%. Other signs of toxicity were not observed at any of the dose levels (up to 300 μg/kg/d) (Bucci and Parker 1991).

D. Discussion

GB is not currently regulated under the Resource Conservation and Recovery Act (RCRA) program by the U.S. Environmental Protection Agency (EPA). However, it is regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. GB content is also treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

GB is an acutely toxic chemical agent contained in stockpiled Defense Department munitions. As such, it may be considered an acutely toxic commercial chemical product.

In accordance with the criteria defined in Section VII.A, the Utah Department of Environmental Quality, Division of Solid and Hazardous Waste (DSHW) proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).
- Redefine as a 40 CFR 261.33(e) commercial chemical product P901 in R315-2-11(e).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.
- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).
- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Because of its rapid hydrolysis on dissolution in water, GB is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14.

2. GD (Soman or O-Pinacolyl Methylphosphonofluoridate) CAS Reg. No.96-64-0

CH_3-P(=O)(O-CH[CH_3][C(CH_3)$_3$])F

A. Background

GD, an acutely toxic, relatively nonpersistent nerve agent, was developed by the Germans during World War II but never used. It was designed to be a somewhat more persistent nerve agent than GB. Subsequently, it was produced in small amounts by the U.S. armed services for laboratory testing but was not stockpiled or employed in combat. GD is not included in any CAIS.

B. Physicochemical Properties

The following pressure-temperature relationship was reported for GD (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 7.47060 - 1{,}903.10/(216.9 + t[°C]).$$

GD's evaporation rate is about 1/4 that of water (HQ/DA,DN,AF 1990). GD's hydrolytic half-life is highest (the rate slowest) in the pH range of 4 to 7, about 144 hours at pH 5 and 20°C, and decreases outside that range in either more alkaline or more acidic solutions (Clark 1989). The observed hydrolysis rate constant (K_{obs}) at 25°C, exclusive of buffer effects (Healy 1948), is as follows:

$$k_{obs}\ (h^{-1}) = 0.0047 + 33[H_3O^+] + 5x10^4[OH^-].$$

The hydrolytic half-life for GD at pH 10 can be estimated as 8.3 minutes (or, accepting the value from Yang et al. 1992, 12 minutes). Thus, in an hour, five or more half-lives would elapse, and the concentration of agent would be no more than 3% of its original value. In 4 hours at pH 10, the concentration of GD would be less than one-millionth of its original value.

The strong catalytic effect of aqueous hypochlorite ion on the hydrolysis of GB (Epstein et al. 1956) has also been demonstrated in the catalyzed hydrolysis of GD (Eskanow 1978). Environmentally relevant data for GD are presented in Table 3. GD is about 2% soluble in water.

It may be concluded that once released to the environment, GD would be expected to persist only for a relatively short period. Moreover, as with GB, GD can be detoxified with mild chemical treatment.

TABLE 3 Environmentally Relevant Properties of GD

Property	Data	Reference
Empirical formula	$C_7H_{16}FO_2P$	Not applicable
Molecular weight (MW), g/mol	182.18	Not applicable
Density, g/mL	1.0223/25°C	Samuel et al. 1983
Melting point, °C	-42	Samuel et al. 1983
Boiling point, °C	197.8	Samuel et al. 1983
Vapor pressure at 25°C, torr	0.40/25°C	Samuel et al. 1983
	0.274/20°C	Estimate from the equation for log P (see text).
Log K_{ow}	1.760	King and Brown 1993
	1.82	Experimental value[a]
Aqueous solubility, g/L	34/0°C	Edgewood Arsenal 1974
	21/20°C	Samuel et al. 1983
K_H, atm·m³/mol	3.1×10^{-6}/20°C	Estimate from the equation for log P (see text).
Log K_{oc}	1.17	Estimate from the equation for log P (see text).
	2.37	Calculated[b]

[a] See Appendix H of Background Document E.

[b] Derived from Log K_{ow} of 1.82, as shown above, using equation from Lyman et al. (1982).

C. Toxicity

GD is the most toxic of the G agents (HQ/DA,DN,AF 1990); the toxicity varies with the route of administration. It is significantly more effective than GB by the dermal route. The LCt_{50} for GD estimated for 70-kg humans breathing at the rate of 15 L/min is about 70 mg·min/m³ (Edgewood Arsenal 1974). If the agent is completely absorbed through the lungs, this value is equivalent to an oral LD_{50} of about 15 μg/kg. Table 4 shows the lethal effects of GD in experimental animals by different routes of administration.

Like all nerve agents, GD exerts its effects through inhibition of the enzyme AChE, which is required for nerve and muscle function. Normally, AChE prevents accumulation of acetylcholine after its release as a chemical messenger in the nervous system. AChE inhibition adversely affects skeletal muscle, parasympathetic end organ, and central nervous system operation. Individuals

TABLE 4 Comparison of Some Toxicities of GD by Different Routes of Administration in Different Species

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Mouse	35 μg/kg
Intravenous	LD_{50}	Rat	44.5 μg/kg
Inhalation	LD_{50}	Mouse	1 mg/m^3/10 min
Subcutaneous	LD_{50}	Guinea pig	24 μg/kg
Subcutaneous	LD_{50}	Rat	75 μg/kg
Subcutaneous	LD_{50}	Rabbit	20 μg/kg
Intraperitoneal	LD_{50}	Rat	117 μg/kg
Intraperitoneal	LD_{50}	Mouse	393 μg/kg

Source: Sweet (1987).

poisoned by sufficient amounts of GD may show the following signs and symptoms soon after exposure (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990):

- Difficulty in breathing, tightness of chest;
- Dimness of vision and pinpointing of the eye pupils;
- Drooling and excessive sweating;
- Nausea;
- Vomiting, cramps, and loss of bladder/bowel control;
- Twitching, jerking, and staggering;
- Headache, confusion, drowsiness, coma, and convulsion; and
- Death.

An important characteristic that distinguishes the behavior of GD from that of certain other nerve agents (e.g., GB) is that ChE inhibited by GD undergoes spontaneous "aging" to an inhibited form of the enzyme that is resistant to either spontaneous or therapeutic reactivation (Daniels 1990).

The number and severity of symptoms of GD poisoning depend on the quantity and route of entry of this nerve agent into the body, as well as the duration of exposure. When the eyes are exposed to agent vapor, a prominent sign is pinpointing of the pupils. Dimness of vision results from the reduced amount of light entering the eyes. However, if exposure to the nerve agent is through the skin or by ingestion, the pupils may be normal or only slightly reduced in size. In this event, diagnosis must rely on other symptoms of nerve agent poisoning (HQ/DA,DN,AF 1990).

Respiratory exposure usually results in the onset of symptoms in 2 to 5 minutes; lethal doses kill in less than 15 minutes. Exposure through the eyes produces a very rapid onset of symptoms (usually in less than 2 to 3 minutes). Liquid agent in the eyes kills nearly as rapidly as respiratory exposure. Symptoms from skin absorption appear more slowly. Death from skin absorption may occur in 1 to 2 hours. Very small skin dosages sometimes cause local sweating and tremors (muscle twitching) but few other effects.

If a victim recovers from acute GD poisoning, the recovery will be complete unless anoxia and convulsions have gone unchecked for so long that irreversible central nervous system damage has occurred (HQ/DA,DN,AF 1990).

Ninety-day subchronic toxicity tests of GD in male rats gave an LOAEL of 0.0175 mg/kg/d (5 days per week, adjusted to 0.0125 mg/kg/d for 7 days per week) of agent administered by gavage (Bucci and Parker 1992). At the end of the first week of exposure, red blood cell cholinesterase was depressed 18%.

D. Discussion

GD is not currently regulated under the RCRA program by EPA. However, it is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. GD content is also considered a basis for listing hazardous waste as F999 [R315-50-9(1)].

GD is an acutely toxic chemical agent, and although it is not contained in stockpiled Defense Department munitions or any CAIS, it has been produced in small quantities for testing purposes. It is not considered a commercial chemical product, and, accordingly, is not proposed to be given a "P" or "U" code. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Because of its rapid hydrolysis on dissolution in water, GD is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14.

3. GA (Tabun or O-Ethyl N,N-Dimethyl Phosphoramidocyanidate) CAS Reg. No. 77-81-6

$C_5H_{11}N_2O_2P$

A. Background

GA, also an acutely toxic, relatively nonpersistent nerve agent, was developed and weaponized by the Germans beginning in 1942 during World War II (Brophy et al. 1959), but it was never used. It has been considered a serious threat agent. Although GA was not produced in the United States, small stocks imported from Germany have been maintained here and used for experimental purposes. GA is not included in any CAIS.

B. Physicochemical Properties

GA's hydrolytic half-life is highest (the rate slowest) in the pH range of 4 to 7, about 8.5 hours at 20°C, decreasing outside that range in either more alkaline or more acidic solutions. The persistence of the agent varies widely with surface (e.g., soil type) and ambient conditions. GA is significantly more persistent than GB (Penski et al. 1992) and persists longer on sand or sandy loam than on concrete (Cooper 1990). It evaporates about 20 times more slowly than water (HQ/DA,DN,AF 1990). For example, when concrete was loaded with GA at 3.33 mg/cm^2 at 25°C and the evaporated GA collected and measured, 99% of that recovered had been emitted in the first 13 hours, whereas the process took only 2 hours for GB (Cooper 1990). Some of the agent decomposed or was trapped in the contaminated medium, so that there was never complete recovery. Some environmentally relevant properties of GA are given in Table 5. In addition to data

TABLE 5 Environmentally Relevant Properties of GA

Property	Data	Reference
Empirical formula	$C_5H_{11}N_2O_2P$	Not applicable
Molecular weight (MW), g/mol	162.13	Not applicable
Density, g/mL	1.0756	Samuel et al. 1983
Melting point, °C	-50	Samuel et al. 1983
Boiling point, °C	247.5	Samuel et al. 1983
Vapor pressure at 25°C, torr	2.057	Samuel et al. 1983
	0.07	Edgewood Arsenal 1974
Log K_{ow}	0.56	Britton and Grant 1988
	0.384	Experimental value[a]
Aqueous solubility, g/L	72/20°C; 98/25°C	Safety Office, undated
K_H, atm·m^3/mol	7.0 x 10^{-7}/25°C	Estimated from the equation for log P (see text).
Log K_{oc}	0.79	Estimated from the equation for log P (see text).
	1.59	Calculated[b]

[a] See Appendix H of Background Document E.

[b] Derived from Log K_{ow} of 0.384, as shown above, using equation from Lyman et al. (1982).

in Table 5, the following pressure-temperature relationship was reported for GA (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 6.80011 - 1{,}700.59/(186.4 + t[°C]).$$

It may be concluded that once released to the environment, GA would be expected to persist for a relatively short period. Moreover, as with GB, GA can be detoxified with a mild chemical treatment.

C. Toxicity

GA is less toxic than GB, GD, or GF (Lewis 1992). The toxicity of this nerve agent varies somewhat, depending on how it is delivered. Table 6 shows the lethal effects of GA in experimental animals by different routes of administration.

The LCt_{50} value estimated for humans breathing at the rate of 15 L/min was 135 mg·min/m^3 (Edgewood Arsenal 1974). If GA is completely absorbed in the lungs and acts systemically, the corresponding LD_{50} would be 0.029 mg/kg.

TABLE 6 Comparison of Some Toxicities of GA by Different Routes of Administration in Different Species

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Mouse[a]	150 μg/kg
Intravenous	LD_{50}	Rat[a]	66 μg/kg
Inhalation	LD_{50}	Mouse[a]	15 mg/m³/30 min
Subcutaneous	LD_{50}	Rat[a]	193 μg/kg
Subcutaneous	LD_{50}	Rabbit[a]	375 μg/kg
Intraperitoneal	LD_{50}	Rat[b]	800 μg/kg (approximate)
Intraperitoneal	LD_{50}	Mouse[a]	420 μg/kg
Oral	LD_{50}	Rat[a]	604 μg/kg

[a] Sweet (1987).

[b] Edgewood Arsenal (1974).

Like all nerve agents, GA exerts its effects through inhibition of the enzyme AChE, which is required for nerve and muscle function. Normally, AChE prevents accumulation of acetylcholine after its release as a chemical messenger in the nervous system. AChE inhibition adversely affects skeletal muscle, parasympathetic end organ, and central nervous system operation. Individuals poisoned by sufficient amounts of GA might show the following signs and symptoms soon after exposure (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990):

- Difficulty in breathing, tightness of chest;
- Dimness of vision and pinpointing of the eye pupils;
- Drooling and excessive sweating;
- Nausea;
- Vomiting, cramps, and loss of bladder/bowel control;
- Twitching, jerking, and staggering;
- Headache, confusion, drowsiness, coma, and convulsion; and
- Death.

The number and severity of symptoms depend on the quantity and route of entry of GA into the body. On ocular exposure to the agent vapor, a prominent sign would be pinpointing of the pupils. Dimness of vision would result from the reduced amount of light entering the eyes. However, if exposure to GA were through the skin or by ingestion, the pupils might be normal or only slightly reduced in size. In this event, diagnosis would have to rely on the symptoms of nerve agent poisoning other than effects on the pupils (HQ/DA,DN,AF 1990).

Respiratory exposure would result in the onset of symptoms in about 2 to 5 minutes; lethal doses would probably kill in less than 15 minutes. Exposure through the eyes would produce a very rapid onset of symptoms (probably less than 2 to 3 min). Liquid in the eyes would kill nearly as rapidly as respiratory exposure. GA, though about half as toxic as GB, is more irritating to the eyes (Edgewood Arsenal 1974). Symptoms would appear more slowly from skin absorption than by inhalation or ocular exposure. If exposure was sufficient, skin absorption might cause death in 1 to 2 hours (HQ/DA,DN,AF 1990).

A 13-week (subchronic) toxicity test of GA was conducted with rats. The no-observed-adverse-effect (NOAEL) was 28.13 µg/kg/d (5 days per week, adjusted to 20.09 µg/kg/d for 7 days per week) of agent administered by intraperitoneal injection (Bucci et al. 1992).

D. Discussion

GA is not currently regulated under the RCRA program by EPA. However, it is regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. GA content is also considered a basis for listing hazardous waste as F999 [R315-50-9(1)].

GA, an acutely toxic chemical agent, is contained in a relatively small Defense Department munitions stockpile (imported from Germany after World War II and kept for experimental purposes). GA is not included in CAIS. GA may be considered an acutely toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).
- Redefine as a 40 CFR 261.33(e) commercial chemical product P902 in R315-2-11(e).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Because of its rapid hydrolysis on dissolution in water, GA is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14.

4. GF (Cyclohexyl Methylphosphonofluoridate) CAS Reg. No. 329-99-7

$C_7H_{14}FO_2P$

A. Background

GF, an acutely toxic, relatively nonpersistent nerve agent, was developed by the Germans during World War II but was never used. It has been considered a serious threat agent. GF is not maintained in the U.S. chemical stockpile and is not included in any CAIS.

B. Physicochemical Properties

The chemistry of GF has been explored less extensively than that of GA, GB, or GD. Although GF has almost the same molecular weight as GD, its evaporation rate is only 1/5 that of the latter (HQ/DA,DN,AF 1990). Environmentally relevant data are shown in Table 7. In addition, GF shows the following pressure-temperature relationship (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 6.56240 - 1{,}407.30/(170.4 + t[°\text{C}]).$$

The behavior of similar G agents indicates that once released to the environment, GF would persist for relatively short periods. Moreover, as with the other G agents, GF can be detoxified with mild chemical treatment.

TABLE 7 Environmentally Relevant Properties of GF

Property	Data	Reference
Empirical formula	$C_7H_{14}FO_2P$	Not applicable
Molecular weight (MW), g/mol	180.18	Not applicable
Density, g/mL	1.1278/25°C	Samuel et al. 1983
Melting point, °C	-30	Samuel et al. 1983
Boiling point, °C	239.0	Samuel et al. 1983
Vapor pressure, torr	0.070/25°C 0.0442/20°C	Samuel et al. 1983 Estimated from the equation for log P (see text).
Log K_{ow}	1.493	King and Brown, 1993
Aqueous solubility, g/L	37/20°C	Samuel et al. 1983
K_H, atm·m^3/mol	2.8×10^{-7}/20°C	Estimated from the equation for log P (see text).
Log K_{oc}	1.82	Estimated from the equation for log P (see text).

C. Toxicity

The information available on acute toxicity of GF appears to be limited to LD_{50} values (expressed in µg/kg) by the subcutaneous route (Sweet 1987): Guinea pig, 100; hamster, 130; mouse, 400; rat, 225; and rabbit, 100.

Like all nerve agents, GF exerts its effects through inhibition of the enzyme AChE, which is required for nerve and muscle function in multicellular animals. Normally, AChE prevents accumulation of acetylcholine after its release as a chemical messenger in the nervous system. AChE inhibition adversely affects skeletal muscle, parasympathetic end organ, and central nervous system operation. Individuals poisoned by GF would show the following symptoms (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990):

- Difficulty in breathing and tightness of chest;
- Dimness of vision and pinpointing of the eye pupils;
- Drooling and excessive sweating;
- Nausea;
- Vomiting, cramps, and loss of bladder/bowel control;

- Twitching, jerking, and staggering;
- Headache, confusion, drowsiness, coma, and convulsion; and
- Death.

The number and severity of symptoms would depend on the quantity and route of entry of GF into the body. When the eyes are exposed to agent vapor, a prominent sign is pinpointing of the pupils. Dimness of vision results from the reduced amount of light entering the eyes. However, if exposure to GF were through the skin or by ingestion, the pupils might be normal or only slightly reduced in size. In this event, diagnosis would have to rely on the symptoms of nerve agent poisoning other than its effects on the pupils (HQ/DA,DN,AF 1990).

Respiratory exposure to agents such as GF usually results in onset of symptoms in 2 to 5 minutes; lethal doses kill in less than 15 minutes. Exposure through the eyes would produce a very rapid onset of symptoms (usually less than 2 to 3 minutes). Liquid in the eyes would kill nearly as rapidly as respiratory exposure. Symptoms should appear more slowly from skin absorption. If exposure was sufficient, skin absorption might cause death in 1 to 2 hours. Very small skin dosages might cause local sweating and tremors but few other effects.

If a victim recovers from acute GF poisoning, the recovery should be complete unless anoxia and convulsions have gone unchecked sufficiently long to cause irreversible central nervous system damage (HQ/DA,DN,AF 1990).

D. Discussion

GF is not currently regulated under the RCRA program by EPA, nor is it regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. GF content is not now considered a basis for listing hazardous waste as F999 [R315-50-9(1)]. DSHW suggested including GF in a draft rule it released to the Army for review in March 1996. Hence, it is evaluated herein.

GF is an acutely toxic chemical agent, and although it is not contained in stockpiled Defense Department munitions or in CAIS, it has been produced in small quantities for testing purposes. It is not considered a commercial chemical product, and, accordingly, it is not proposed to be given a "P" or "U" code. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Because of its rapid hydrolysis on dissolution in water, GF is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14.

5. VX [O-Ethyl S-(2-Diisopropylaminoethyl) Methylphosphonothioate] CAS Reg. No. 50782-69-9

CH_3-P(=O)(OC_2H_5)(S-CH_2-CH_2-N[CH(CH_3)$_2$]$_2$)

A. Background

Following World War II, the British developed the nerve agent VX to circumvent respiratory protection, which is fairly effective against other nerve agents, namely G agents. VX was subsequently adopted by the United States, produced in quantity, tested in laboratories and in the open air, stored in 1-ton containers, and loaded in munitions that were stockpiled but never employed in combat. VX is not contained in any CAIS.

VX, which is dispersed as an aerosol because of its low volatility, is acutely toxic, highly effective by the percutaneous route (100 times as effective as GB [HQ/DA,DN,AF 1990]), and although more persistent than the G agents, is nevertheless relatively nonpersistent.

B. Physicochemical Properties

The following pressure-temperature relationship was reported for VX (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 7.28100 - 2{,}072.10/(172.5 + t[°C]).$$

VX, a good skin penetrant, is more persistent than G agents because of its low vapor pressure — its evaporation rate is about 1/1,500 that of water (HQ/DA,DN,AF l990). The pK_a of protonated VX at 25°C has been given as 8.60 (Epstein et al. 1974) or as 9.1 (Demek et al. 1970).

VX hydrolysis rates (Table 8) tend to be slower (half-life longer) than those of the G agents; thus, at pH 10 and 25°C, the half-life of VX in water is 2,432 minutes (converted data from Epstein et al. [1974]), compared with 4½ minutes for GB. At pH 5 and 25°C, the half-life was reported as 2,342 hours (Clark 1989).

The hydrolytic reactions of VX can involve multiple pathways. Both rates and products depend on pH (Epstein et al. 1974; Yang et al. 1990, 1992, 1993, 1994; Szafraniec et al. 1993), temperature, and VX concentration (Yang et al. 1994). VX is not subject to acid-catalyzed hydrolysis but does undergo water- and hydroxyl-ion-catalyzed hydrolysis.

Studies in the Netherlands on the degradation of VX in soil involved application of 200 mg/kg of VX to soil (Verweij and Boter 1976; Kaaijk and Frijlink 1977). After three weeks, only 0.1% of the applied VX was detectable. Thus, it may be concluded that VX is not chemically stable in soil.

TABLE 8 Hydrolysis Half-Lives for VX

pH	Half-Life (hours) @ 25°C
2.0	2,520
4.0	2,257
6.0	2,381
7.0	996
8.0	184
9.0	63
10.0	40.5
11.0	15
12.0[a]	2.5
12.65	0.525
12.9	0.279
13.5	0.0529

[a] Approximately 0.01 M NaOH.

Source: Epstein et al. (1974).

The chemistry of decontamination of VX with chlorine bleaches is complex, but VX is effectively detoxified by these oxidants (Yang et al. 1992, 1994). Environmentally relevant physicochemical properties of VX are presented in Table 9.

Once released to the environment, VX would be expected to persist for moderate periods (weeks). The most effective way to detoxify VX would be with hypochlorite-containing bleach.

It may be concluded that VX would persist in the environment for longer periods than G agents, but would decompose completely in a matter of weeks.

C. Toxicity

As with the G agents, VX exerts its physiologic effects through inhibition of the enzyme AChE. VX is especially effective percutaneously. Individuals poisoned

TABLE 9 Environmentally Relevant Properties of VX

Property	Data	Reference
Empirical formula	$C_{11}H_{26}NO_2PS$	Not applicable
Molecular weight (MW), g/mol	267.38	Not applicable
Density, g/mL	1.0083/25°C	Samuel et al. 1983
Melting point, °C	-50	Samuel et al. 1983
Boiling point, °C	298.4	Samuel et al. 1983
Vapor pressure, torr	6.2×10^{-4}/25°C	Samuel et al. 1983
Log K_{ow}	2.09 (estimate)	Small 1984
Aqueous solubility, g/L	30/25°C	Edgewood Arsenal 1974
K_H, atm·m³/mol	7.2×10^{-9}/25°C	Estimated from the equation for log P (see text).
Log K_{oc}	1.18	Sage and Howard 1989

by ingestion of VX may show the following signs and symptoms (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990):

- Difficulty in breathing and tightness of chest;
- Dimness of vision and pinpointing of the eye pupils;
- Drooling and excessive sweating;
- Nausea, vomiting, cramps, and loss of bladder/bowel control;
- Twitching, jerking, and staggering;
- Headache, confusion, drowsiness, coma, and convulsion; and
- Death.

The LCt_{50} value for VX, estimated for humans breathing at the rate of 15 L/min, is 30 mg·min/m³ (Edgewood Arsenal 1974). If the compound is completely absorbed, the equivalent oral LD_{50} would be 0.0064 mg/kg. According to Michel et al. (1962), the rat oral LD_{50} for VX is 0.178 mg/kg.

Because VX has such a low volatility, liquid droplets on the skin do not evaporate as do uncovered droplets of GB; thus, effective percutaneous absorption can take place. Hence, by this route, VX is estimated to be more than 100 times as toxic as GB to man (Edgewood Arsenal 1974).

If a victim recovers from acute VX poisoning, the recovery will be complete unless anoxia and convulsions have gone unchecked long enough to cause irreversible central nervous system damage (HQ/DA,DN,AF 1990).

In a 90-day (subchronic) toxicity test conducted for VX with rats, the LOAEL was 0.25 µg/kg/d (5 days per week, adjusted to 0.179 µg/kg/d for 7 days per week) of agent administered by subcutaneous injection. The only adverse effect at this dosage was a decrease of the red blood cell cholinesterase level to a low of 33% in males at 60 days and 48% in females at 30 days (Goldman et al. 1988).

D. Discussion

VX is not currently regulated under the RCRA program by the EPA. However, it is regulated under the current State of Utah P999 hazardous waste code [R315-2-11(e)]. VX content is also treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

VX is an acutely toxic chemical agent and is contained in the Defense Department munitions stockpile, although it is not included in any CAIS. As such, VX may be considered an acutely toxic commercial chemical product. In accordance with the criteria defined in Section VII.A, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Redefine as a 40 CFR 261.33(e) commercial chemical product P903 in R315-2-11(e).

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Because of its fairly rapid hydrolysis on dissolution in water, VX is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14. VX is more persistent than the G agents but, relatively speaking, is not significantly persistent.

6. HD/H [Sulfur Mustard or Bis(2-Chloroethyl) Sulfide] CAS Reg. No. 505-60-2

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

A. Background

Mustard gas (HD/H, H is undistilled mustard; HD refers to distilled mustard) was first synthesized in 1822, but its toxicity was only discovered in 1860. Germany first used H militarily during World War I (July 1917) in Belgium; it was soon adopted, produced in large amount, and employed by the Allied powers (Rosenblatt et al. 1975). H and HD were produced and stockpiled by both sides during World War II, but they were not used in that conflict. HD or H is contained in some CAIS.

H differs from HD in that H contains certain impurities normally absent from HD; however, there is no standard composition for H. Production grade H (manufactured by the Levinstein process) was a mixture of mustard with sulfur and various impurities (Fuson et al. 1946). The H was distilled to improve the agent's storage stability, separating HD from a sulfur-rich waste sludge. Virtually all available physicochemical information on what is commonly known as "mustard gas" has been determined on the relatively pure HD.

B. Physicochemical Properties

Table 10 lists environmentally relevant properties of HD. In addition to the data in Table 10, the following pressure-temperature relationship was reported for HD (Penski 1993):

$$\log P\ (\text{torr}) = 7.4749753 - 1{,}940.711/(204.6712 + t[°C]).$$

HD has a low solubility in water and a low rate of solution. For these reasons, HD is difficult to decontaminate by aqueous hydrolysis despite the relatively high first-order rate constant of the reaction once the HD is in solution.

Despite the rapidity with which the hydrolysis reaction occurs, there is a tendency, in quiescent conditions, for HD to polymerize at the HD/water interface, interfering with transfer of HD to the aqueous phase and thus further shielding the bulk agent from hydrolysis reactions (MacNaughton and Brewer 1994). However, with adequate mixing, HD can be detoxified.

TABLE 10 Environmentally Relevant Properties of HD

Property	Data	Reference
Empirical formula	$C_4H_8Cl_2S$	Not applicable
Molecular weight (MW), g/mol	159.08	Not applicable
Density, g/mL	1.2685/25°C	Samuel et al. 1983
Melting point, °C	14.445	Penski 1993
Boiling point, °C	217.5	Samuel et al. 1983
Vapor pressure, torr	0.082/22°C[a]	Samuel et al. 1983
	0.1059/25°C[a]	Samuel et al. 1983
Log K_{ow}	1.37	EPA 1986
Aqueous solubility, g/L	0.92/22°C	Edgewood Arsenal 1974
K_H, atm·m³/mol	2.57 x 10^{-5}	Sage and Howard 1989
Log K_{oc}	2.0-2.1	Sage and Howard 1989

[a] Calculated from the equation for log P (see text).

Small (1984) cited work by Puzderliski (1980) in an example of the rate of environmental disappearance of HD under relatively unfavorable conditions. HD in soil that was exposed to light rain at 0°C would disappear by a factor of 1,500 in three months. At 25°C, the time for this to occur was about 2 days.

The sulfur moiety of HD is readily subject to decontamination by oxidation with various forms of hypochlorite-containing materials, such as bleach solution (approximately 5% aqueous sodium hypochlorite), chlorinated lime, or "high-test hypochlorite" (HTH) (Yang et al. 1992).

If released to the environment in low concentrations, HD would not be expected to persist for long, but sizable masses could remain, more or less encapsulated, for considerable periods. HD can be effectively decontaminated with hypochlorite.

C. Toxicity

HD is a vesicant (blister agent), as well as an alkylating agent producing cytotoxic effects on the hematopoietic (blood-forming) tissues. Its primary effects are on the skin and eyes, although it is also toxic by inhalation or ingestion (Edgewood Arsenal 1974). Table 11 shows the lethal effects of HD in experimental animals by different routes of administration.

TABLE 11 Comparison of Some Toxicities of HD by Different Routes in Different Species

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Mouse	8,600 μg/kg
Intravenous	LD_{50}	Rat	700 μg/kg
Inhalation	LD_{50}	Mouse	120 mg/m^3/20 min
Subcutaneous	LD_{50}	Rat	1,500 μg/kg
Subcutaneous	LD_{50}	Rabbit	20,000 μg/kg
Subcutaneous	LD_{50}	Mouse	20,000 μg/kg

Source: Sweet (1987).

The human respiratory LCt_{50} for HD is 1,500 mg·min/m^3 (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990). The percutaneous LCt_{50} is estimated as 10,000 mg·min/m^3 (HQ/DA,DN,AF 1990). The estimated human oral LD_{50} for HD is 0.7 mg/kg (Edgewood Arsenal 1974); this estimate seems low in view of the intragastric LD_{50} of 17 mg/kg for rats (Edgewood Arsenal 1974). The human percutaneous LD_{50} is estimated as 100 mg/kg (Safety Office undated). For percutaneously exposed (but masked) personnel, an air Ct (concentration x time) of as little as 1,000 mg·min/m^3 can be incapacitating (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990). The human respiratory ICt_{50} (concentration x time that is incapacitating to 50% of a population) for this agent is 150 mg·min/m^3 (HQ/DA,DN,AF 1990). Eye injury from HD occurs at an air Ct of about 100 mg·min/m^3 (Edgewood Arsenal 1974). Effects of HD are not immediately manifest; the latent period for appearance of ocular symptoms is 4-12 hours after mild exposure, 3-6 hours after moderate exposure, and 1-3 hours after severe exposure (Edgewood Arsenal 1974).

In acute (single-episode) exposures to HD vapors, the eyes are especially sensitive. Symptoms of mild exposure (after a latent period) consist of tearing and a sensation of "sand" in the eyes, with the conjunctiva and lids becoming swollen and fluid-filled. Higher exposures bring on blepharospasm (inability to keep the eyes open), blurring of vision, mucoserous discharge, and other symptoms of ocular irritation. At the highest exposures, there is also headache, deep ocular pain, ulceration, necrosis, and dense corneal opacification. Convalescence can take as long as several months (Edgewood Arsenal 1974).

Reaction to acute skin exposure to HD is first manifested (after some delay) by gradual reddening of the skin, as if by sunburn, accompanied by itching and mild burning. This is followed by blistering; pinpoint lesions form, enlarge, and coalesce to large translucent yellowish blisters. If the blisters do not rupture, they are resorbed in about a week. Mustard burns of the skin are usually followed by a persistent brown pigmentation except at the site of vesication, which may be depigmented. In addition to surface effects, high levels of HD vapor would be absorbed through the skin into the body to cause systemic effects. These effects may include anorexia, nausea, vomiting, depression, and fever (Edgewood Arsenal 1974).

Respiratory tract lesions caused by acute exposure to HD develop slowly over several days. Symptoms begin with hoarseness, which may progress to aphonia (loss of voice), cough, fever, and dyspnea (shortness of breath). Incidence of bronchopneumonia is high. Convalescence is slow, and coughing may persist for a month or longer (Edgewood Arsenal 1974).

Ingestion of food or water contaminated with liquid HD produces nausea, vomiting, pain, diarrhea, and prostration (Edgewood Arsenal 1974).

The eyes of workers exposed to HD for over 2 months showed low-grade conjunctival infection, reduced corneal sensitivity, and staining or pigmentation of the corneal epithelium (Edgewood Arsenal 1974).

Repeated skin exposure may lead to hypersensitivity of the skin to HD. Sensitization is followed by a more rapid onset of symptoms upon re-exposure to low levels of HD, as well as by the development of a dermatitis similar to that from poison ivy (Edgewood Arsenal 1974). Exposures of munitions plant workers to HD for 3 weeks to 6 months led these men to seek treatment for respiratory distress. Typically, males developed some or all of the following symptoms: red eyes, photophobia, lacrimation, impaired vision, blepharospasm, loss of taste and smell, nose bleed, sore throat, chest pain, wheezing, and dyspnea. Furthermore, repeated exposure to HD led to lingering bronchitis, bronchial asthma, hoarseness, aphonia, and hypersensitivity to smoke, dust, and fumes (Panel on Cholinesterase Reactivator Chemicals et al. 1984).

HD is a carcinogen via inhalation in animals and humans, as well as a mutagen (Panel on Cholinesterase Reactivator Chemicals et al. 1984). An upper bound (95% confidence limit) unit inhalation cancer risk of 8.5×10^{-2} µg/m^3 has been calculated for HD (Koppikar et al. 1991).

In a 21-week study of rats, HD was dissolved in sesame oil and administered by gavage (stomach tube), giving an LOAEL of 0.022 mg/kg-d. (This average level was obtained by dividing the total dose of HD at the lowest dose level by 147 days.) The critical effect was epithelial hyperplasia of the forestomach, an anatomical feature not present in humans (Sasser et al. 1989a). A chronic toxicity criterion for HD is presented in Section VII.D of the Preamble.

D. Discussion

HD is currently listed by EPA as a 40 CFR Part 261, Appendix VIII hazardous constituent. 40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, "Hazardous Constituents." HD is the only chemical agent proposed in this regulation that is specifically listed by EPA in Appendix VIII.[2] HD is not included by EPA as an acutely toxic or toxic commercial chemical product, however.

H and HD (which essentially refer to the same chemical) are currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. HD content is also currently treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

HD is an acutely toxic chemical agent and is contained in the Defense Department munitions stockpile, as well as in some CAIS. As such, HD may be considered an acutely toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).
- Redefine as a 40 CFR 261.33(e) commercial chemical product P904 in R315-2-11(e).
- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).
- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

HD is also proposed to be added to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent. Although already listed in EPA's 40 CFR 261, Appendix VIII, which is adopted by the state in R315-50-10, DSHW is proposing to list this chemical in R315-50-10 for reasons of continuity and consistency.

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[2] HN2 is also specifically listed by the EPA in 40 CFR 261, Appendix VIII; however, this chemical is not proposed for listing in the currently proposed rule (see Background Document C).

HD dissolves very slowly in water. Because of its rapid hydrolysis once dissolved in (or mixed with) water, HD is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

7. L (Lewisite 1 or 2-Chlorovinyldichloroarsine) CAS Reg. No. 541-25-3

$ClCH=CH\text{-}AsCl_2$

A. Background

Lewisite (L or L1), although known in an impure state since 1904, was first characterized by Professor W. Lee Lewis of Northwestern University in 1918, too late to be employed as a vesicant (blister) agent in World War I. It was manufactured during World War II and stored in 1-ton containers (Rosenblatt et al. 1975). L is therefore part of the U.S. stockpile and is also included in some CAIS.

Plant-run L is a complex mixture containing the *cis* and *trans* isomers of L, *bis*(2-chlorovinyl)chloroarsine (lewisite 2) and *tris*(2-chlorovinyl)arsine (lewisite 3), along with traces of metal catalyst. Lewisite 2 and 3 are always present in lesser concentrations in formulations of lewisite 1; conversely, if lewisite 1 is not present, neither lewisite 2 nor 3 will be either. In all cases, results of analyses for lewisite 1 will serve as indicators of the presence of lewisite 2 and 3 as well. Lewisite 2 and 3 are discussed in Sections 16 and 17, respectively.

B. Physicochemical Properties

L has been separated into the *cis* and *trans* forms, some properties of which have been determined (Whiting 1948). Table 12 lists environmentally relevant properties for the production-grade *cis trans* mixture. In addition, the following pressure-temperature relationship has been reported for L (Samuel et al. 1983):

$$\log P\ (\text{torr}) = 6.40361 - 1{,}237.03/(155.2 + t[°C]).$$

C. Toxicity

The human LCt_{50} for L via inhalation has been estimated (Safety Office undated) at 1,200-1,500 mg·min/m^3, which, for a breathing rate of 0.015 m^3/min, would amount to an LD_{50} of 0.26-0.32 mg/kg for this route. The human LCt_{50} for exposure to L vapor via the skin only is reckoned at a higher level, 100,000 mg·min/m^3 (Safety Office undated). The intraocular ICt_{50} is $<$300 mg·min/m^3 (Safety Office undated). In animals, the LD_{50} of L (presumably

TABLE 12 Environmentally Relevant Properties of L

Property	Data	Reference
Empirical formula	$C_2H_2AsCl_3$	Not applicable
Molecular weight (MW), g/mol	207.35	Not applicable
Density, g/mL	1.8793/25·C	Samuel et al. 1983
Melting point, °C	-18 ± 0.1	Edgewood Arsenal 1974
Boiling point, °C	195.9	Samuel et al. 1983
Vapor pressure, torr	0.35/25°C	Samuel et al. 1983
	0.58	McNaughton and Brewer 1994
	1.56	Edgewood Arsenal 1974
Aqueous solubility, g/L	0.5	Beilstein Sys. No. 410[a]

[a] Because L is so rapidly hydrolyzed on contact with water, there is, in essence, no meaning to this water-related constant.

as the liquid) via the dermal route is invariably higher than via the subcutaneous route. For example, in the rat, the values are 24 mg/kg and 1 mg/kg, respectively (Goldman and Dacre 1989). Curiously, the rat oral LD_{50} is reported by Goldman and Dacre (1989) as 50 mg/kg, a value even higher than the skin LD_{50}, and odd when compared with another reported rat oral LD_{50} of 5 mg/kg (Lewis 1992).

Table 13 compares the lethal effects of L in experimental animals by different routes of administration.

Unlike sulfur mustard, for which the effects can be delayed for several hours, L irritates skin immediately on contact and is instantly irritating to the eyes. (It is also toxic on inhalation.) The burning sensation on the skin gradually increases, but remains bearable (if exposure is not too extensive); the skin becomes red, and blisters form. Healing can take up to a few weeks (Goldman and Dacre 1989). Serious injury to the eyes by L vapor may sometimes be avoided, because of the almost immediate and searing sensations, which cause the eyelids to close and alert the victim to the need for precautionary measures. Such measures include timely administration of the antidote, British antilewisite (BAL)(Goldman and Dacre 1989).

The systemic effects of L include pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, low blood pressure, interference with kidney function, and hemoconcentration caused by loss of fluid from the bloodstream. In nonfatal cases, hemolysis of erythrocytes has occurred, with a resultant hemolytic

TABLE 13 Comparison of Some Toxicities of L by Different Routes of Administration in Different Species

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Rabbit	500 μg/kg
Inhalation	LC_{50}	Mouse	500 mg/m^3/9 min
Subcutaneous	LD_{50}	Guinea pig	1,000 μg/kg
Subcutaneous	LD_{50}	Rat	1,000 μg/kg
Subcutaneous	LD_{50}	Rabbit	2,000 μg/kg
Skin	LD_{50}	Rat	15,000 μg/kg
Skin	LD_{50}	Rabbit	4,000 μg/kg

Source: Sweet (1987).

anemia. Excretion of oxidized products into the bile by the liver produces focal necrosis of the liver, necrosis of the mucosa of the biliary passages with periobiliary hemorrhages, and some injury to the intestinal mucosa (Edgewood Arsenal 1974; Safety Office undated; Goldman and Dacre 1989). The statement has been made (HQ/DA,DN,AF 1990) that, "when humidity is high, L hydrolyzes so rapidly that it is difficult to maintain a concentration sufficient to blister bare skin." Indeed, the fact that L's systemic toxicity is considerably lower via the dermal route than by inhalation, intravenous injection, or subcutaneous injection suggests that a large portion of L is hydrolyzed rapidly and may act locally but does not reach the circulatory system.

Sasser et al. (1989b) conducted a 90-day subchronic toxicity study of L in rats (10 male, 10 female per dose group). They found a NOAEL of 0.5 mg/kg administered 5 days per week.

Repeated exposure to L can cause sensitization and chronic lung impairment (Safety Office undated). Despite statements to the contrary (e.g., in the Material Safety Data Sheets [Safety Office undated]), there is no evidence that L, or any other organic arsenical, would be carcinogenic, mutagenic, or teratogenic (Goldman and Dacre 1989).

The toxicity of L may be attributed to an increase in capillary permeability to plasma proteins. That effect, in turn, appears to be due to the ability of L to interfere with such enzymes as pyruvate oxidase by binding with enzyme thiol groups (Goldman and Dacre 1989).

D. Discussion

Arsenicals (i.e., arsenic-containing chemical agents and associated compounds), including L, are included in the EPA's 40 CFR, Part 261, Appendix VIII, under the general notation "Arsenic Compounds N.O.S. (not otherwise specified)" (40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, "Hazardous Constituents.") L is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. L content is also treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

L is a lethal arsenical chemical agent, a vesicant with rapid toxic manifestations. L is found in some Defense Department stockpiles and is present in some CAIS. As such, L may be considered an acutely toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Redefine as a 40 CFR 261.33(e) commercial chemical product P905 in R315-2-11(e).

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Note also that arsenic-containing wastes not specifically listed as hazardous wastes may be classified as hazardous waste under the EPA toxicity characteristic (incorporated under DSHW regulation R315-2-9) as D004 if it leaches arsenic at levels greater than or equal to 5 mg/L.

Because of its rapid hydrolysis once dissolved in water, L is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14. Arsenic, a component of L, is an EPA 40 CFR 264, Appendix IX, (incorporated under R315-50-14) groundwater monitoring constituent.

8. HN1 [Bis(2-Chloroethyl)ethylamine] CAS Reg. No. 538-07-8

$(ClCH_2\text{-}CH_2\text{-})_2N\text{-}CH_2\text{-}CH_3$

A. Background

HN1 is one of a series of nitrogen analogs of sulfur mustard with vesicant properties that were first noted in 1935. A large number of such nitrogen mustards were synthesized and investigated during World War II, when pilot quantities of HN1 were made. About 100 tons of HN1 was produced in the United States in 1943 (Brophy et al. 1959). Perhaps because of poor storage stability, none of the nitrogen mustards was standardized as a chemical agent. Nitrogen mustards (specifically HN2, discussed in Background Document C) are used in cancer chemotherapy, but HN1 apparently is not currently applied for this purpose. HN1 is included in some CAIS.

B. Physicochemical Properties

HN1 polymerizes slowly in munitions (HQ/DA,DN,AF 1990). Because of its low solubility in water, HN1 hydrolyzes slowly (HQ/DA,DN,AF 1990), but mixing should increase the rate of hydrolysis. Table 14 lists environmentally relevant properties of HN1.

C. Toxicity

The physiological effects of nitrogen mustards are similar to those of sulfur mustard (HD), but nitrogen mustards are somewhat more effective against the eyes than HD. As with HD, nitrogen mustards cause delayed vesicancy. Table 15 shows the lethal effects of HN1 in experimental animals by different routes of administration.

It appears that only acute toxicities have been determined for HN1. However, HN1 is a mutagen and suspected of being a carcinogen.

TABLE 14 Environmentally Relevant Properties of HN1

Property	Data
Empirical formula	$C_6H_{13}Cl_2N$
Molecular weight (MW), g/mol	170.1
Density, g/mL	1.09/25°C
Melting point, °C	-34
Boiling point, °C	194[a]
Vapor pressure at 25°C, torr	0.24
Aqueous solubility, g/L	Very low

[a] Calculated; decomposes.

Source: HQ/DA,DN,AF (1990).

TABLE 15 Comparison of Some Toxicities of HN1 by Different Routes of Administration in Rats and Mice

Route	Type of Exposure	Species	Toxicity
Intravenous	LD_{50}	Rat	500 µg/kg
Inhalation	LD_{50}	Mouse	900 mg/m^3/10 min
Subcutaneous	LD_{50}	Mouse	1,100 µg/kg
Intraperitoneal	LD_{50}	Mouse	1,030 µg/kg
Oral	LD_{50}	Rat	2,500 µg/kg

Source: Sweet (1987).

D. Discussion

HN1 is not currently regulated under the RCRA program by EPA. However, it is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. HN1 content is also treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

HN1, like HD, is a lethal chemical agent, a vesicant with delayed toxic manifestations. Within the domain of the U.S. government, other than, perhaps, in laboratory quantities, HN1 is found in CAIS, but not in stockpiled munitions. HN1 may be considered an acutely toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).
- Redefine as a 40 CFR 261.33(e) commercial chemical product P906 in R315-2-11(e).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.
- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

HN1 dissolves very slowly in water; once dissolved in water it should hydrolyze rapidly, and, therefore, it is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

9. HN3 [Tris(2-Chloroethyl)amine] CAS Reg. No. 55-77-1

$(ClCH_2\text{-}CH_2\text{-})_3N$

A. Background

HN3 is one of a series of nitrogen analogs of sulfur mustard with vesicant properties that were first noted in 1935. A large number of such nitrogen mustards were synthesized and investigated during World War II, when the Germans prepared considerable stocks of munitions filled with HN3 and the British made small quantities of HN3 on a pilot scale. Because of its history, as well as because it is the most stable of the three nitrogen mustards, HN3 is the principal representative of those three compounds (HQ/DA,DN,AF 1990). Nitrogen mustards (specifically HN2, discussed in Background Document C) are used in cancer chemotherapy, but HN3 apparently is not currently used for this purpose. HN3 is included in some CAIS.

B. Physicochemical Properties

HN3 is sufficiently stable to be used in munitions even under tropical conditions; however, the agent darkens and deposits a crystalline solid on storage (HQ/DA,DN,AF 1990). At high concentrations, HN3 hydrolyzes slowly in water (because of its low solubility) (HQ/DA,DN,AF 1990). HN3 is more persistent than HD (HQ/DA,DN,AF 1990). In contact with water, HN3 is not completely hydrolyzed after standing for days (Edgewood Arsenal 1974). Table 16 lists environmentally relevant properties of HN3.

TABLE 16 Environmentally Relevant Properties of HN3

Property	Data
Empirical formula	$C_6H_{12}Cl_3N$
Molecular weight (MW), g/mol	204.5
Density, g/mL	1.24/25°C
Melting point, °C	-3.7
Boiling point, °C	256[a]
Vapor pressure at 25°C, torr	0.0109
Aqueous solubility, g/L	0.08

[a] Calculated; decomposes.

Sources: HQ/DA,DN,AF (1990); Edgewood Arsenal (1974).

C. Toxicity

The physiological effects of nitrogen mustards are similar to those of sulfur mustard (HD), but nitrogen mustards are somewhat more effective against the eyes than HD (HQ/DA,DN,AF 1990). HN3 is a vesicant similar to HD; it produces eye injury, damage to the respiratory tract, and, after absorption into the body, cytotoxic actions in a variety of tissues. The hematopoietic and lymphoid tissues are especially sensitive. These cytotoxic effects follow absorption through the intact skin, respiratory, or gastrointestinal tracts (Edgewood Arsenal 1974).

As stated above, HN3 is about as potent a vesicant as HD. Like HD, HN3 causes delayed skin blistering, although ocular effects may be immediate. Table 17 shows the lethal effects of HN3 in rats and mice by different routes of administration. It appears that only acute toxicities have been determined for HN3. However, HN3 is a mutagen and suspected of being a carcinogen.

TABLE 17 Comparison of Some Toxicities of HN3 by Different Routes in Rats and Mice

Route	Type of Exposure	Species	Toxicity
Inhalation	LC_{50}	Mouse	120 mg/m^3/10 min
Subcutaneous	LC_{50}	Mouse	6,900 μg/kg
Subcutaneous	LC_{50}	Rat	2,000 μg/kg
Oral	LC_{50}	Rat	5,000 μg/kg

Source: Sweet (1987).

D. Discussion

HN3 is not currently regulated under the RCRA program by EPA. However, it is regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. HN3 content is also treated as a basis for listing hazardous waste as F999 [R315-50-9(1)].

HN3, like HD, is a lethal chemical agent, a vesicant with delayed toxic manifestations. Within the domain of the U.S. government, other than, perhaps, in laboratory specimens, HN3 is found in CAIS, but not in stockpiled munitions. HN3 may be considered an acutely toxic commercial chemical product.

In accordance with the criteria defined in Section VII.A, the DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Redefine as a 40 CFR 261.33(e) commercial chemical product P907 in R315-2-11(e).

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

HN3 dissolves very slowly in water, but once dissolved, it should hydrolyze rapidly. For this reason, HN3 is not proposed for inclusion as a 40 CFR 264 Appendix IX groundwater monitoring constituent in R315-50-14.

10. HL [Mustard (HD)-Lewisite (L) Mixture]
CAS Reg. No. Not Applicable

$$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl \quad + \quad ClCH=CH\text{-}AsCl_2$$

A. Background

The usual HL mixture is 37% HD and 63% L by weight. Pure HD freezes at 14.4°C and pure L freezes at -18°C; this eutectic mixture (plant grade material) freezes at about -42°C. Other mixtures may be used with a higher content of the more potent HD if the temperatures at which the mixtures are to be employed are not too low (HQ/DA,DN,AF 1990). The eutectic mixture may be more suitable for use in extremely cold weather. HL has not been stockpiled or tested by the Defense Department, and it has not been used as a munitions fill. HL is not included in any CAIS.

B. Physicochemical Properties

The following temperature/pressure (torr) relationships have been calculated for the mixture (HQ/DA,DN,AF 1990): -10°C/0.02; 20°C/0.248; 40°C/1.03. The liquid density at 20°C is 1.66 g/mL.

C. Toxicity

The main military purpose for this mixture is to deliver liquid HD to the target; thus, effects of the L may be considered of secondary importance. Skin contamination produces immediate stinging, although blister formation is delayed about 13 hours. Liquid HL causes severe damage to the eyes. Except in severe cases, respiratory damage is similar to that produced by HD alone (HQ/DA,DN,AF 1990).

D. Discussion

HL is not currently regulated under the RCRA program by EPA, although HD is currently listed by EPA as a 40 CFR Part 261, Appendix VIII, hazardous constituent, and L is included in Appendix VIII through the listing of arsenic compounds not otherwise specified (40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, "Hazardous Constituents"). HL is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. HL content is also considered as a basis for listing hazardous waste as F999 [R315-50-9(1)].

HL is a mixture of the aforementioned acutely toxic chemical agents, HD and L, with vesicant and eye irritant — immediate and delayed — toxic manifestations. However, HL is not known to be contained in the U.S. stockpile or in CAIS.

DSHW notes that its current listing for HL as a P999 commercial chemical product is in deviation from standard EPA practice, where the "P" and "U" codes refer only to commercial chemical products in which the chemical is the sole active ingredient. Because HL is a mixture of two active ingredients, it would not be included in EPA's rules as a commercial chemical product. As indicated in Section VII.A of the Preamble, however, DSHW proposes to continue its practice of regulating mixtures as commercial chemical products even though they consist of two active ingredients. Hence, HL is proposed as a commercial chemical product, despite recognition that it is not EPA practice to do so. Although HL is not in the U.S. chemical stockpile nor in any CAIS, DSHW is proposing HL as a commercial chemical product because it contains HD.

In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Redefine as a 40 CFR 261.33(e) commercial chemical product P908 in R315-2-11(e).

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

DSHW is not proposing to add HL to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent or to 40 CFR 261, Appendix VII, as a basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1). The individual compounds HD and L are proposed to be included in these lists.

HD dissolves very slowly in water; but once dissolved in water, both HD and L, are very rapidly hydrolyzed. For this reason, HL is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

11. HQ [Mustard (HD)-Q-Mustard (Q) Mixture]
CAS Reg. No. Not Applicable

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

+

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

A. Background

The usual HQ mixture is 75% HD and 25% Q (Safety Office 1995b). (Q-mustard is discussed in Section 14.) HQ has not been stockpiled in the United States or tested by the Defense Department, and it has not been used as a munitions fill. It is also not present in any CAIS.

B. Physicochemical Properties

The vapor pressure of HQ is calculated as 0.088 torr at 25°C (slightly less than that of HD)(Safety Office 1995a); the equilibrium vapor phase should be almost entirely HD. HQ should be barely soluble in water and should dissolve very slowly. Once dissolved, however, it should hydrolyze fairly rapidly. Like HD, HQ can be readily decontaminated with various hypochlorite mixtures (Safety Office 1995a).

C. Toxicity

Both HD and Q are vesicants (blister agents), as well as alkylating agents that produce cytotoxic effects on the hematopoietic (blood-forming) tissues. Their primary effects are on the skin and eyes, although they are also toxic by inhalation or ingestion (Edgewood Arsenal 1974). It may be assumed that the effects of the mixture are delayed. Ten-minute inhalation toxicities for Q vs. HD are compared in Table 18 for five mammal species.

TABLE 18 Inhalation Toxicity of Q and HD in Some Mammalian Species

	LC_{50} (mg/m^3/10 min)	
Species	Q	HD
Cat	900	70
Guinea pig	8	200
Mouse	6	120
Rat	11	100
Rabbit	2,000	280

Source: Sweet (1987).

D. Discussion

HQ is not currently regulated under the RCRA program by EPA, although HD is currently listed by EPA as a 40 CFR Part 261, Appendix VIII, hazardous constituent (40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, "Hazardous Constituents"). HQ is not regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)], and HQ content is not considered as a basis for listing hazardous waste as F999 [R315-50-9(1)]. DSHW suggested including HQ in a draft rule it released to the Army for review in March 1996. Hence, it is evaluated herein.

HQ is a mixture of acutely toxic chemical agents, HD and Q, with vesicant and eye irritant (mainly delayed) toxic manifestations. HQ is not known to be contained in the U.S. stockpile or in any CAIS.

DSHW notes that listing HQ as a P999 commercial chemical product would be a deviation from standard EPA practice, where the "P" and "U" codes refer only to commercial chemical products in which an individual chemical is the sole active ingredient. Because HQ is a mixture of two active ingredients, it would not be included under EPA rules as a commercial chemical product. However, as indicated in Section VII.A of the Preamble, and as proposed for HL, DSHW proposes to continue with its listing practice. DSHW proposes to regulate mixtures as commercial chemical products even though they consist of two active ingredients. Hence, HQ is proposed as a commercial chemical product, despite recognition that it is contrary to EPA practice to do so. Although HQ is not in the U.S. chemical stockpile and is not in any CAIS, DSHW is proposing HQ as a commercial chemical product because it contains HD.

In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

- Define as a 40 CFR 261.33(e) commercial chemical product P909 in R315-2-11(e).

DSHW is not proposing to add HQ to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent or to 40 CFR 261, Appendix VII, as a basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1). The individual compounds HD and Q are proposed to be included in these lists.

HD dissolves very slowly in water, but once dissolved, both HD and Q are very rapidly hydrolyzed. For this reason, HQ is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

12. HT [Mustard (HD)-T-Mustard (T) Mixture]
CAS Reg. No. Not Applicable

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$
+
$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}O\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

A. Background

The usual HT mixture is 60% HD and 40% T by weight (T-mustard is discussed in Section 15). This mixture can be prepared directly from relatively nontoxic starting materials. Pure HD freezes at 14.4°C and pure T freezes at 9.0°C (Samuel 1983), while the 60/40 eutectic mixture freezes at 0°C to 1.3°C (HQ/DA,DN,AF 1990). The mixture is said to be more stable and more persistent than HD (HQ/DA,DN,AF 1990). The U.S. military stockpile includes HT in munitions and ton containers, but HT is not present in any CAIS.

B. Physicochemical Properties

The vapor pressure of HT is 0.104 torr, and the density 1.269 g/mL at 25°C (HQ/DA,DN,AF 1990). The mixture boils above 228°C. It is barely soluble in water, dissolving very slowly. However, once dissolved, it hydrolyzes fairly rapidly. Like HD, HT can be decontaminated with various hypochlorite mixtures (HQ/DA,DN,AF 1990).

C. Toxicity

The main military reason for using the HT mixture is to deliver liquid HD to the target at temperatures below the freezing point of that compound. HT skin contamination produces strong, but delayed, blistering, as well as eye irritation (HQ/DA,DN,AF 1990).

D. Discussion

HT is not currently regulated under the RCRA program by EPA, although HD is currently listed by EPA as a 40 CFR 261, Appendix VIII, hazardous constituent (40 CFR Part 261, Appendix VIII, is adopted by the State of Utah as R315-50-10, "Hazardous Constituents"). HT is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. HT content is also considered as a basis for listing hazardous waste F999 [R315-50-9(1)].

HT is a mixture of lethal chemical agents and a vesicant and eye irritant with some immediate and some delayed toxic manifestations. It is stockpiled in Defense Department munitions and ton containers, but it is not in any CAIS.

DSHW notes that its current listing for HT as a P999 commercial chemical product is in deviation from standard EPA practice, where the "P" and "U" codes refer only to commercial chemical products in which an individual chemical is the sole active ingredient. Because HT is a mixture of two active ingredients, it would not be included in EPA rules as a commercial chemical product. As indicated in Section VII.A of the Preamble, however, DSHW proposes to continue with its listing practice. DSHW proposes to regulate mixtures as commercial chemical products even though they consist of two active ingredients. Hence, HT is proposed as a commercial chemical product, despite recognition that it is contrary to EPA practice to do so. Although HT is in the U.S. chemical stockpile, it is not in any CAIS. DSHW is proposing HT as a commercial chemical product because it contains HD and also because HT is in the U.S. stockpile in Utah.

In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).

- Redefine as a 40 CFR 261.33(e) commercial chemical product P910 in R315-2-11(e).

- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).

DSHW is not proposing to add HT to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent or to 40 CFR 261, Appendix VII, as a basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1). The individual compounds HD and T are proposed to be included in these lists.

HD dissolves very slowly in water; both HD and T, having once dissolved in water, are very rapidly hydrolyzed. For this reason, HT is not proposed for inclusion as a 40 CFR 264 Appendix IX, groundwater monitoring constituent in R315-50-14.

13. DM (Adamsite or Phenylarsazine Chloride) CAS Reg. No. 578-94-9

$C_{12}H_9AsClN$

A. Background

DM, an arsenical, was developed as a sternutator (vomiting compound) by the United States at the end of World War I. DM is not in the U.S. stockpile, but it is included in some CAIS. It is disseminated as an aerosol and is effective only through the respiratory tract; it has no irritant effect on the skin (HQ/DA,DN,AF 1990).

B. Physicochemical Properties

DM is a relatively persistent agent of extremely low vapor pressure (Edgewood Arsenal 1974). It hydrolyzes slowly, and evidently reversibly, in water because the presence of HCl in the water prevents significant hydrolysis. Additional information on environmentally relevant properties of DM is given in Table 19.

C. Toxicity

DM is a vomiting agent of relatively moderate toxicity. Intravenous LD_{50} values for the mouse and the rabbit are, respectively, 35,000 µg/kg and 6,000 µg/kg (Sweet 1987).

TABLE 19 Environmentally Relevant Properties of DM

Property	Data	Reference
Empirical formula	$C_{12}H_9AsClN$	NA
Molecular weight (MW), g/mol	277.57	NA
Density, g/mL	1.65/20°C	Edgewood Arsenal 1974
Melting point, °C	195	Edgewood Arsenal 1974
Boiling point, °C	410[a]	HQ/DA,DN,AF 1990
Vapor pressure at 25°C, torr	4.5×10^{-11}	Edgewood Arsenal 1974
Aqueous solubility, g/L	0.064 (room temp.)	Edgewood Arsenal 1974
K_H, atm°m³/mol	2.6×10^{-13}/25°C	Estimated from the equation for log P (see text).

[a] *Calculated, decomposes.*

The human inhalation LCt_{50} has been given as 11,000 mg·min/m³ (e.g., 1,000 mg/m³ for 11 minutes). The human ICt_{50} is said to be 22 to 150 mg·min/m³. Both the rate of action (onset in about 1 minute) and detoxification (often within about 30 minutes after incapacitation) are rapid (HQ/DA,DN,AF 1990).

Sternutators such as DM produce strong, pepper-like irritation in the upper respiratory tract, with irritation of the eyes and mucous membranes, tearing, and changes in the function of the salivary glands. Sternutators cause violent, uncontrollable sneezing, coughing, severe headache, acute pain, tightness in the chest, nausea, vomiting, and a general feeling of bodily discomfort, depression, and malaise. When released indoors, they can cause serious illness, even death (HQ/DA,DN,AF 1990).

Specific information on the carcinogenicity of DM is lacking, but the World Health Organization is cited (Goldman and Dacre 1989) as saying that "there is no conclusive evidence that any of the organoarsenic compounds tested for carcinogenicity in laboratory animals are carcinogenic."

D. Discussion

DM is not currently listed by EPA, although arsenicals (arsenic-containing chemical agents and associated compounds), including DM, are incorporated into EPA's 40 CFR, Part 261, Appendix VIII, under the general notation, "Arsenic

Compounds N.O.S. (not otherwise specified)." (40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, Hazardous Constituents.)

DM is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)], nor is DM content treated as a basis for listing hazardous waste F999 [R315-50-9(1)]. The Army suggested that DM be considered for listing as an acutely toxic ("P" code) or toxic ("U" code) commercial chemical product. Hence, it is evaluated herein.

DM was developed by the United States during World War I as an incapacitating, rather than lethal, agent. Within the domain of the U.S. government, other than in laboratory specimens, DM is found in some CAIS but not in stockpiled munitions. DM may be considered a toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

- Define as a 40 CFR 261.33(e) commercial chemical product U901 in R315-2-11(e).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.
- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Note also that arsenic-containing wastes not specifically listed as hazardous wastes may be classified as hazardous waste under the EPA toxicity characteristic (incorporated under DSHW regulation R315-2-9) as D004 if it leaches arsenic at levels greater than or equal to 5 mg/L.

Because the solubility of DM is believed to be low, it is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14. In any event, arsenic, a component of DM, is already a 40 CFR 264 Appendix IX (incorporated under R315-50-14) groundwater monitoring constituent.

14. Q [Q-Mustard or Sesquimustard or 1,2-Bis(2-Chloroethylthio)ethane] CAS Reg. No. 3563-36-8

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

A. Background

Q is a mustard-like vesicant (blister agent). It has been reported as a minor constituent (<5%) of both H and HD (Rosenblatt et al. 1996). Q "has been identified as the most powerful vesicant known" (Safety Office 1995b). Q does not appear to have been deliberately made for use alone, but has been evaluated as a mixture with HD (see Section 11). It is, therefore, not present in the U.S. stockpile nor in any CAIS.

B. Physicochemical Properties

Vapor pressures of Q have been reported for various temperatures, as follows (Safety Office 1995b).

t (°C)	P (torr)
90	0.02
110	0.15
140	2.00
181	15.0

Hence, the following regression equation can be developed for Q of ln P vs. T (K):

$$\ln P\ (\text{torr}) = 29.5679 - 12{,}092.16/T\ (\text{K}).$$

Table 20 lists environmentally relevant properties of Q. It may be concluded that Q is far less volatile than HD and represents no vapor threat, only a possible contact threat.

C. Toxicity

Mustard-like compounds such as Q are vesicants (blister agents), as well as alkylating agents that produce cytotoxic effects on the hematopoietic (blood-forming) tissues. Their primary effects are on the skin and eyes, although they are also toxic by inhalation or ingestion (Edgewood Arsenal 1974).

The inhalation toxicity of Q has been measured in six mammalian species for the same time period (Table 21).

TABLE 20 Environmentally Relevant Properties of Q

Property	Data	Reference
Empirical formula	$C_6H_{12}Cl_2S_2$	Not applicable
Molecular weight (MW), g/mol	219.19	Not applicable
Density, g/mL	1.272/25°C	Safety Office 1995b
Melting point, °C	56-57	Safety Office 1995b
Boiling point, °C	254	Estimate[a]
Vapor pressure, torr, 25°C	8.0×10^{-6}	Estimate[b]
	3.5×10^{-5}	Safety Office 1995b
Log K_{ow}	1.54	Estimate[a]
Aqueous solubility, g/L	0.025/25°C	Safety Office 1995b
K_H, atm·m³/mol	9.2×10^{-8}	Estimate[a]
Log K_{oc}	1.6	Estimate[a]

[a] Estimated by linear regression (ln P = A-B/T with data in the text).

[b] Estimated using linear regression (see footnote a) and correction for solid state, assuming a rigid molecule.

D. Discussion

Q is not currently regulated under the RCRA program by EPA. Furthermore, it is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. Neither is Q content treated as a basis for listing hazardous waste as F999 [R315-50-9(1)]. However, DSHW suggested including Q in a draft rule it released to the Army for review in February 1996. Hence, it is evaluated herein.

Q is not in the U.S. stockpile, other than as a minor constituent of HD, and is not in any CAIS. Because Q is not in the stockpile or in any CAIS, it should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however, because it can be present in HD formulations. In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

TABLE 21 Inhalation Toxicity, LC_{50}, of Q in Some Mammalian Species

Species	LC_{50} (mg/m³/10 min)
Cat	900
Guinea pig	8
Hamster	22
Mouse	6
Rat	11
Rabbit	2,000

Source: Sweet (1987).

- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Since Q would always be only a minor constituent of HD, Q is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

15. T [T-Mustard or Bis(2-Chloroethylthioethyl) ether] CAS Reg. No. 63918-89-8

$Cl\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}O\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}Cl$

A. Background

T is a mustard-like vesicant (blister agent). It is apparently not a normal constituent of either H or HD (Rosenblatt et al. 1996). It has either been manufactured for the purpose of mixing it with HD to produce the vesicant agent HT (see Section 12) or was generated as a co-product with HD by modifying the HD synthetic process. When it exists, T is always found with HD. T is in the U.S. stockpile, but only as a component of HT. T is not present in any CAIS.

B. Physicochemical Properties

The following pressure-temperature relationship was reported (Samuel et al. 1983) for liquid T:

$$\log P \text{ (torr)} = 9.53000 - 4{,}191.00/T \text{ (K)}.$$

Table 22 contains a list of environmentally relevant properties for T. It may be concluded that T is considerably less volatile than HD and represents no vapor threat, only a possible contact threat.

C. Toxicity

Mustard-like compounds such as T are vesicants (blister agents), as well as alkylating agents that produce cytotoxic effects on the hematopoietic (blood-forming) tissues. Their primary effects are on the skin and eyes, although they are

TABLE 22 Environmentally Relevant Properties of T

Property	Data	Reference
Empirical formula	$C8H_{16}Cl_2S_2$	Not applicable
Molecular weight (MW), g/mol	263.25	Not applicable
Density, g/mL	1.2362/25°C	Samuel et al. 1983
Melting point, °C	8.97	Samuel et al. 1983
Boiling point, °C	357	Samuel et al. 1983
Vapor pressure, torr	3.0×10^{-5}	Samuel et al. 1983
Log K_{ow}	0.68	Estimated from the equation for log P (see text).
Aqueous solubility, g/L	Practically insoluble	Safety Office updated

also toxic by inhalation or ingestion (Edgewood Arsenal 1974). The inhalation LC_{50} for T in mice is 1,650 mg/m^3/10M (Sweet 1987).

D. Discussion

T is not currently regulated under the RCRA program by EPA; however, it is currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. T content is also treated as a basis for listing hazardous waste F999 [R315-50-9(1)].

Pure T is not in the U.S. military stockpile, but it is present as a major constituent of the agent HT, which is in the stockpile. T is not present in any CAIS. Hence, T should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Remove current 40 CFR 261.33(e) P999 designation in R315-2-11(e).
- Remove from 40 CFR 261 Appendix VII basis for listing hazardous waste F999 in R315-2-10(e) and R315-50-9(1).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.

- Add as a 40 CFR 261 Appendix VII basis for listing newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Since T would always occur in admixture with HD, T is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

16. Lewisite 2 [Bis(2-Chlorovinyl)chloroarsine] CAS Reg. No.40334-69-8

$(ClCH{=}CH\text{-})_2AsCl$

A. Background

Lewisite 2 is an impurity of L (lewisite 1). The proportion of lewisite 2 in plant-run L depends on the efficacy of process control (Rosenblatt et al. 1975). Lewisite 2 is not in the U.S. stockpile and is not included in any CAIS, other than as an impurity of L.

B. Physicochemical Properties

A colorless or pale yellow liquid, lewisite 2 is "insoluble" in water and dilute acids. Lewisite 2 is less volatile than L (Rosenblatt et al. 1975). Table 23 lists environmentally relevant properties for lewisite 2. In addition, the following pressure-temperature relationship has been reported for lewisite 2 (Jackson and Jackson 1935):

$$\log P \text{ (torr)} = 9.983 - 3295.3/T \text{ (K)}.$$

TABLE 23 Environmentally Relevant Properties of Lewisite 2

Property	Data	Reference
Empirical formula	$C_4H_4AsCl_3$	Not applicable
Molecular weight (MW), g/mol	233.36	Not applicable
Density, g/mL	1.6926/24.1°C	Jackson and Jackson 1935
Melting point, °C	Liquid	Jackson and Jackson 1935
Boiling point, °C	230	Jackson and Jackson 1935
Vapor pressure, torr	0.085/25°C	Estimated from the equation for log P (see text).
Aqueous solubility, g/L	"Insoluble"	Jackson and Jackson 1935

C. Toxicity

The acute toxicity of lewisite 2 is comparable to that of L. LD_{50} values for guinea pigs are 200 µg/kg by the subcutaneous route and 8,000 µg/kg via the skin (Sweet 1987). Applied to the skin, lewisite 2 has vesicating properties but is much less powerful in this respect than L; its irritant properties on the respiratory system, however, are more intense than those of L (Jackson and Jackson 1935).

D. Discussion

Arsenicals (i.e., arsenic-containing chemical agents and associated compounds), including L and L2, are incorporated into EPA's 40 CFR, Part 261, Appendix VIII, under the general notation "Arsenic Compounds N.O.S. (not otherwise specified)." (40 CFR Part 261, Appendix VIII, is adopted by the State of Utah as R315-50-10, "Hazardous Constituents.") Lewisite 2 is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)], and Lewisite 2 content is not treated as a basis for listing hazardous waste as F999 [R315-50-9(1)]. However, DSHW suggested including lewisite 2 in a draft rule it released to the Army for review in February 1996. Hence, it is evaluated herein.

Lewisite 2 is not in the U.S. military stockpile, other than as a minor constituent of L, and is not in any CAIS. Because lewisite 2 is not in the stockpile, it should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent.

- Add as a 40 CFR 261, Appendix VII, basis for listing newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Note also that arsenic-containing wastes not specifically listed as hazardous wastes may be classified as hazardous waste under the EPA toxicity characteristic (incorporated under DSHW regulation R315-2-9) as D004 if it leaches arsenic at levels greater than or equal to 5 mg/L.

Since lewisite 2 would always be only a minor constituent of L, lewisite 2 is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14. Arsenic is already a 40 CFR 264, Appendix IX, (incorporated under R315-50-14) groundwater monitoring constituent.

17. Lewisite 3 [Tris(2-Chlorovinyl)arsine]
CAS Reg. No.40334-70-1

$(ClCH{=}CH\text{-})_3As$

A. Background

Like lewisite 2, lewisite 3 is an impurity of lewisite 1 (L). The proportion of lewisite 3 in plant-run L depends on the efficacy of process control (Rosenblatt et al. 1975). Lewisite 3 is not in the U.S. stockpile and is not included in any CAIS, other than as an impurity of L.

B. Physicochemical Properties

A colorless, low-melting solid, lewisite 3 is "insoluble" in water, dilute acids, and alcohol (Jackson and Jackson 1935). Lewisite 3 is less volatile than L (Rosenblatt et al., 1975). Table 24 lists environmentally relevant properties for lewisite 3. In addition, the following pressure-temperature relationship has been reported for lewisite 3 (Jackson and Jackson 1935):

$$\log P\ (\text{torr}) = 9.159 - 3312.43/T\ (\text{K}).$$

C. Toxicity

The toxicity of lewisite 3 is significantly less than that of L (Rosenblatt et al. 1975). This conclusion would agree with the probable mechanism of action of L, which requires the presence of a thiol-displaceable chlorine on the arsenic. Lewisite 3 is neither a strong vesicant nor a powerful respiratory irritant; however,

TABLE 24 Environmentally Relevant Properties of Lewisite 3

Property	Data	Reference
Empirical formula	$C_6H_6AsCl_3$	Not applicable
Molecular weight (MW), g/mol	259.38	Not applicable
Density, g/mL	1.5664/23.7°C	Jackson and Jackson 1935
Melting point, °C	23 or less	Jackson and Jackson 1935
Boiling point, °C	260	Jackson and Jackson 1935
Vapor pressure, torr	0.011/25°C	Estimated from the equation for log P (see text).
Aqueous solubility, g/L	"Insoluble"	Jackson and Jackson 1935

its odor is pungent and most unpleasant and induces violent sneezing (Jackson and Jackson 1935).

D. Discussion

Arsenicals (i.e., arsenic-containing chemical agents and associated compounds), including L and L3, are included into EPA's 40 CFR, Part 261, Appendix VIII, under the general notation, "Arsenic Compounds N.O.S. (not otherwise specified)." (40 CFR Part 261, Appendix VIII is adopted by the State of Utah as R315-50-10, "Hazardous Constituents.") Lewisite 3 is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. Lewisite 3 content is not treated as a basis for listing hazardous waste as F999 [R315-50-9(1)]. However, DSHW suggested including lewisite 2 in a draft rule it released to the Army for review in February 1996. Hence, it is evaluated herein.

Lewisite 3 is not in the U.S. military stockpile and is not in any CAIS, other than as a minor constituent of L. Because lewisite 3 is not in the stockpile and not in any CAIS, it should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent.

- Add as a 40 CFR 261, Appendix VII, basis for listing newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Note also that arsenic-containing wastes not specifically listed as hazardous wastes may be classified as such under the EPA toxicity characteristic (incorporated under DSHW regulation R315-2-9) as D004 if it leaches arsenic at levels greater than or equal to 5 mg/L.

Since it would always be only a minor constituent of L, lewisite 3 is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14. Arsenic is already a 40 CFR 264 Appendix IX (incorporated under R315-50-14) groundwater monitoring constituent.

18. EA2192 [S-(2-Diisopropylaminoethyl) Methylphosphonothioic Acid] CAS Reg. No. 73207-98-4

$CH_3P(=O)(S\text{-}CH_2\text{-}CH_2\text{-}N[CH\{CH_3\}_2]_2)(OH)$

A. Background

EA2192 is one of several primary hydrolysis products when VX undergoes hydrolysis, with or without hydroxide ion catalysis. EA2192 exists only as a potential breakdown product of VX.

B. Physicochemical Properties

Among the main differences between EA2192 and the parent VX are their physicochemical properties. EA2192 is about 3,700 times as stable as VX toward alkaline hydrolysis in 1 M sodium hydroxide (Yang et al. 1993). It is completely stable in distilled water for at least 1,000 hours (Kingery and Allen 1995). The higher (ammonia) 18 pK_a of EA2192 is cited by Kingery and Allen (1995) as 11.2 and by Michel et al. (1962) as 10.6. The lower (phosphonate) pK_a was reported as 0.6 (Michel et al. 1962). The compound doubtless exists as a dipolar ion (zwitterion) in neutral aqueous solution and must be hydrophilic (Rosenblatt 1996). Its vapor pressure, and especially its Henry's Law constant, K_H, should be considerably lower than those of VX (Rosenblatt 1996). EA2192 is formed when VX decomposes in soil, but its rate of disappearance in that medium is evidently greater than that of VX (Kingery and Allen 1995). It may be concluded that EA2192 could be persistent in the aqueous environment, although probably not in soil.

Table 25 lists some environmentally relevant properties for EA2192.

C. Toxicity

EA2192, like its parent VX, is a cholinesterase inhibitor. EA2192 has a mean intravenous LD_{50} in two species of 0.015 mg/kg (Safety Office undated), while VX has a median subcutaneous LD_{50} in four species of 0.014 mg/kg (Sweet 1987); thus, the assertion that EA2192 is almost as toxic as VX (Sage and Howard 1989) is appropriate. A chronic toxicity criterion for EA2192 is derived in Section VII.D of the Preamble.

TABLE 25 Environmentally Relevant Properties of EA2192

Property	Data
Empirical formula	$C_9H_{22}NO_2PS$
Molecular weight (MW), g/mol	239.32
Melting point, °C	138-140

Source: Rosenblatt (1996).

D. Discussion

EA2192 is not currently regulated under the RCRA program by EPA. Furthermore, it is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. EA2192 content is also not presented as a basis for listing hazardous waste as F999 [R315-50-9(1)]. The Army suggested that EA2192 be considered for listing as a hazardous constituent. Hence, it is evaluated herein.

EA2192 is not a chemical agent and is not in the U.S. military stockpile or in any CAIS, other than as a hydrolysis product (impurity) of VX. Because EA2192 is only a breakdown product of VX, it should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent.
- Add as a 40 CFR 261, Appendix VII, basis for listing newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Furthermore, despite considerable uncertainty as to its mobility in groundwater, EA2192 could be proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14. However, because there is already a good indicator (ethyl methylphosphonic acid [EMPA]) for VX that should be easier to monitor (see Section 19), EA2192 is not proposed as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

19. EMPA (Ethyl Methylphosphonic Acid) CAS Reg. No. 1832-53-7

$CH_3P(=O)(OC_2H_5)(OH)$

A. Background

EMPA is a breakdown product of VX. Some EMPA is always produced when VX is hydrolyzed, and this compound occurs in no other context. The presence of EMPA in water is presumptive evidence for the current or former presence of VX.

B. Physicochemical Properties

The properties of EMPA have not been determined, but it is expected to behave similarly to isopropyl methylphosphonic acid (IMPA) (Section 22) and to have a similar environmental fate. The empirical formula of EMPA is $C_3H_9O_3P$, and the molecular weight is 124.075.

C. Toxicity

It is reasonable to assume that EMPA has about the same low toxicity as its homolog, IMPA (Section 22), but no specific data are available on EMPA toxicity. EMPA is expected to be relatively nontoxic.

D. Discussion

EMPA is not currently regulated by EPA under the RCRA program, nor is EMPA currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. EMPA content is not currently considered a basis for listing hazardous waste F999 [R315-50-9(1)]. The Army suggested that the status of EMPA be considered because it is being addressed pursuant to Dugway Proving Ground's installation restoration program. Hence, it is evaluated herein.

EMPA is expected to have a very low toxicity. EMPA is not a chemical agent and is not in the U.S. military stockpile or in any CAIS, other than as a minor hydrolysis product (impurity) of VX. In view of the fact that EMPA is only a breakdown product of VX, it should not be considered as a commercial chemical product. Moreover, considering EMPA's expected low toxicity, it should not be classified as a hazardous constituent. However, EMPA could be a useful groundwater monitoring constituent.

In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14, as an indicator of releases of VX.

20. TDG (Thiodiglycol)
CAS Reg. No. 111-48-8

$HO\text{-}CH_2\text{-}CH_2\text{-}S\text{-}CH_2\text{-}CH_2\text{-}OH$

A. Background

TDG is the principal product when HD is hydrolyzed in the presence of excess water. Moreover, TDG has been used in private industry — in particular for the manufacture of ballpoint pen inks (Eckler 1996) — and is, therefore, an article of commerce. Because of its association with HD, it is subject to export restrictions.

B. Physicochemical Properties

TDG, the major hydrolysis product of HD, is polar. Although it does not undergo further hydrolysis, TDG is subject to environmental biotransformation (Nemeth 1989). Two bacterial strains isolated from "local soil" (presumably at the Edgewood Area of Aberdeen Proving Ground) were able to utilize thiodiglycol as a sole source of carbon for growth. These were Pseudomonas pickettii strain SH18 and Alkaligenes xylosoxidans ssp. xylosoxidans strain SH42. In a period of 72 hours, the SH42 culture metabolized at least 97% of the organic starting material (hydrolyzed HD) to inorganic products. In the case of the SH18 culture, there was a 16% production of thiodiglycol sulfoxide, a product of "dead end" metabolism (Harvey et al. 1992).

Table 26 lists environmentally relevant properties for TDG. As an extremely water-soluble compound of low lipid solubility, TDG would not be expected to bioaccumulate.

C. Toxicity

What little is known of the biological effects of TDG indicates a very low toxicity. The oral LD_{50} values for rats and guinea pigs are 6,610 mg/kg and 3,960 mg/kg, respectively, with relatively steep dose-response curves (Sweet 1987; ICF KE/Clement 1991; Rosenblatt et al. 1975). The intravenous rabbit LD_{50} is 3,000 mg/kg; subcutaneous LD_{50} values in rats and mice are 4,000 mg/kg.

D. Discussion

TDG is not currently regulated by EPA under the RCRA program and is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. TDG content is not currently considered a basis for listing hazardous waste

TABLE 26 Environmentally Relevant Properties of TDG

Property	Data	Reference
Empirical formula	$C_4H_8O_2S$	Not applicable
Molecular weight (MW), g/mol	122.19	Not applicable
Density, g/mL	1.22/20°C	Rosenblatt et al. 1975
Melting point, °C	-10	Rosenblatt et al. 1975
Boiling point, °C	280	Nemeth 1989
Vapor pressure, torr	20/164°C	Rosenblatt et al. 1975
Log K_{ow}	-0.08	Major 1989
Aqueous solubility, g/L	Infinitely miscible	Nemeth 1989

as F999 [R315-50-9(1)]. The Army suggested that the status of TDG be reviewed because it is being addressed pursuant to Dugway Proving Ground's installation restoration program. Hence, it is evaluated herein.

TDG exhibits a very low toxicity. TDG is not a chemical agent and is not in the U.S. military stockpile or in any CAIS, other than as a minor component of HD. Inasmuch as TDG is only a breakdown product of HD, it should not be considered as a commercial chemical product. In view of TDG's expected low toxicity, it should not be classified as a hazardous constituent. However, TDG could be a useful groundwater monitoring constituent. In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

- Add as a 40 CFR 264 Appendix IX ground-water monitoring constituent in R315-50-14, as an indicator of releases of HD.

21. MPA (Methylphosphonic Acid) CAS Reg. No. 993-13-5

$CH_3P(=O)(OH)_2$

A. Background

GB, IMPA, GD, GF, VX, EA2192, EMPA, and some nontoxic agent simulants can all undergo immediate or eventual hydrolysis to the corresponding alkyl- or fluoromethylphosphonate, which may then be hydrolyzed to MPA. The phosphorus-containing nerve agents and their simulants, as well as other related compounds (except for GA and its breakdown products), may all eventually be hydrolyzed to MPA.

B. Physicochemical Properties

MPA is polar. Although it does not undergo chemical hydrolysis and is in general very stable to chemical attack, MPA is subject to environmental biotransformation. Thus, it may undergo slow metabolism to inorganic phosphate by aerobic microorganisms such as *Agrobacter radiobacter* (Kingery and Allen 1995).

MPA has two dissociable protons, with pK_a values of 2.38 and 7.74, respectively (Rosenblatt et al. 1975). MPA is stated to be very soluble in water (Williams et al. 1987). Table 27 lists environmentally relevant properties for MPA.

C. Toxicity

According to Williams et al. (1987), a Material Data Safety Sheet prepared by Thiokol/Ventron Division in 1980 states, "MPA is a skin and eye irritant which may be toxic by skin absorption, ingestion, or inhalation." However, preliminary data indicate acute oral toxicities in rats and mice of 5,000 mg/kg or more.

D. Discussion

MPA is not currently regulated by the EPA under the RCRA program and is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]; neither is MPA content considered a basis for listing hazardous waste as F999 [R315-50-9(1)]. The Army suggested that the status of MPA be reviewed because it is being addressed pursuant to Dugway Proving Ground's installation restoration program. Hence, it is evaluated herein.

MPA exhibits a very low toxicity. It is not a chemical agent and is not in the U.S. military stockpile or in any CAIS, other than as a minor breakdown product of phosphorus-containing agents. Because MPA is only a nerve agent breakdown product, it should not be considered a commercial chemical product. Considering MPA's low toxicity, it should not be classified as a hazardous constituent.

TABLE 27 Environmentally Relevant Properties of MPA

Property	Data	Reference
Empirical formula	CH_5O_3P	Not applicable
Molecular weight (MW), g/mol	96.0	Not applicable
Melting point, °C	107-107.5	Rosenblatt et al. 1975
Aqueous solubility, g/L	Very soluble	Williams et al. 1987

However, MPA could be a useful groundwater monitoring constituent. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14, as an indicator of releases of most phosphorus-containing agents.

22. IMPA (Isopropyl Methylphosphonic Acid) CAS Reg. No. 6838-93-3 (sodium salt) or 1832-54-8 (free acid)

$CH_3P(=O)(OCH[CH_3]_2)(OH)$

A. Background

IMPA is the initial hydrolysis product of GB. As such, it indicates the possible presence of a release of GB into the environment.

B. Physicochemical Properties

IMPA is a colorless liquid that arises from the facile hydrolysis of the nerve agent GB. It may be presumed that this compound is highly water soluble. The pK_a of IMPA was determined to be 2.41; the K_{ow} observable at around pH 7 would be small because the compound would be almost entirely in the anionic form (Major 1989). Kingery and Allen (1994) have estimated a half-life in water at pH 4 and 25°C of 9,100 years. Tests on five soils at ambient temperature (Table 28) showed very diverse hydrolytic half-lives.

TABLE 28 Half-Lives of IMPA in Various Soils[a]

Soil	Half-Life
Dugway Proving Ground sandy clay loam	Infinite
Rocky Mountain Arsenal sandy loam	Infinite
Lakewood, N.J., sand	58 hours
Woodston sandy loam (Gunpowder State Park, Md.)	40 hours
Fort McClellan clay loam	2.9 hours

[a] 2 g soil, 50 mL of 4 mg/L IMPA solution for each test.

Source: Kingery and Allen (1994).

IMPA is biodegraded (hydrolyzed) very slowly by certain microorganisms, apparently nitrifying bacteria; they would not significantly affect the rates of reaction in the soil systems (Kingery and Allen 1994).

Table 29 lists environmentally relevant properties for MPA.

C. Toxicity

There is no evidence that IMPA is particularly toxic. Acute oral LD_{50} values in mice and rats ranged from 5,620 mg/kg in male mice to 7,650 mg/kg in male rats. The mice exhibited soft or liquid stools, reduced motor activity, ataxia, and prostration. On the basis of no-observed-adverse-effect-levels at the highest administered dose in a 13-week study in rats, a reference dose of 100 µg/kg/day, a drinking water equivalent level (DWEL) of 4 mg/L and a lifetime drinking water health advisory value of 700 µg/L were established by the EPA (Roberts and Hartley 1992).

D. Discussion

IMPA is not currently regulated by EPA under the RCRA program. Furthermore, IMPA is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)], and IMPA content is not considered a basis for listing hazardous waste F999 [R315-50-9(1)]. The Army suggested that the status of IMPA be considered because it is being addressed pursuant to Dugway Proving Ground's installation restoration program. Hence, it is evaluated herein.

IMPA exhibits a very low toxicity. It is not a chemical agent and is not in the U.S. military stockpile or in any CAIS, other than as a minor breakdown product of GB. Considering that IMPA is only a breakdown product, it should not be treated as a commercial chemical product. Furthermore, because IMPA's

TABLE 29 Environmentally Relevant Properties of IMPA

Property	Data	Reference
Empirical formula	$C_4H_{11}O_3P$	Not applicable
Molecular weight (MW), g/mol	138.10	Not applicable
Density, g/mL	1.1091/20°C	Rosenblatt et al. 1975
Melting point, °C	Liquid	Rosenblatt et al. 1975
Boiling point, °C	97-98/0.08 torr 123-125/0.2 torr	Rosenblatt et al. 1975
Log K_d (CH_2Cl_2/H_2O)	-2.6	Rosenblatt et al. 1975

toxicity is so low, it should not be classified as a hazardous constituent. However, IMPA could be a useful groundwater monitoring constituent. In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

- Add as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14, as an indicator of releases of GB.

23. LO (Lewisite Oxide, 2-Chlorovinylarsenous Acid) CAS Reg. No. 333-25-5 and 3088-37-7

$C_2H_4AsClO_2$

A. Background

Lewisite oxide (LO) is an environmental breakdown product of L (lewisite 1). This chemical is not present in the U.S. stockpile or in any CAIS, other than as a minor constituents of lewisite 1.

B. Physicochemical Properties

When *cis-trans* Lewisite hydrolyzes (a very rapid reaction [Rosenblatt et al. 1975]), it is first converted to a *cis-trans* gem-diol mixture, Cl-CH=CH-As(OH)$_2$, which slowly loses water to form the *cis* and *trans* Cl-CH=CH-As=O. The *trans*-form of Cl-CH=CH-As=O (but not the *cis*), slowly polymerizes to (Cl-CH=CH-AsO)$_x$ (Rosenblatt et al. 1975). Collectively, these three chemicals are often referred to as lewisite oxide or LO. Because the ratio of these forms is not fixed, varying with time and conditions, the environmentally relevant properties of LO given in Table 30 apply only approximately to LO that may be present as a residue

TABLE 30 Environmentally Relevant Properties of LO

Property	Data	Reference
Empirical formula	$C_2H_4AsClO_2$ (gem-diol)	Not applicable
	C_2H_2AsClO (arsenoxide)	Not applicable
Molecular weight (MW), g/mol	170.45 (gem-diol)	Not applicable
	152.45 (arsenoxide)	Not applicable
Melting point, °C	131 (*cis*)[a]	Hewet 1948
	143 (*trans*)[a]	Hewet 1948
	140 (*trans* polymer)	Rosenblatt et al. 1975
Log K_{ow}	-0.07	Major 1989
Aqueous Solubility, g/L	~10	Rosenblatt et al. 1975

[a] Arsenoxide form.

from long-past chemical operations. These properties are most closely related to the unpolymerized *cis* and *trans* arsenoxide forms of Cl-CH=CH-As=O. The chemical name for Cl-CH=CH-As=O is 2-chlorovinylarsenous oxide. Its chemical formula is C_2H_2AsClO, and its CAS Reg. No. Is 3088-37-7.

LO can slowly hydrolyze to 2-chlorovinylarsonous acid (CVAA) in aqueous media. The CAS Reg. No. For CVAA is 85090-33-1. Its chemical formula is $C_2H_4AsClO_2$ and its molecular weight is 186.43. Under relatively mild conditions, CVAA is oxidized to C1-CH=CH-As (=O) $(OH)_2$, 2-chlorovinylarsonic acid. The empirical formula for 2-chlorovinylarsonic acid is $C_2H_4AsClO_3$, and the CAS Reg. No. Is 64038-44-4. Because the hydrated form of LO is CVAA, analyses in aqueous matrices would focus on CVAA. CVAA may be expected to revert back to LO under drying conditions.

C. Toxicity

No information is available to indicate how irritating or damaging LO would be to the eyes, or how rapidly any damage would occur, and whether intraocular exposure is a reasonable pathway for systemic penetration by LO. It is reasonable to assume that LO absorbed by inhalation or ingestion of dust would have systemic effects similar to those of L, although the pulmonary effects might be less severe than those from inhalation of L.

Any comparison between L and LO must take into account the higher volatility of L. Furthermore, the hydrolysis of L, which might occur within moist tissue, would generate hydrochloric acid at the site. While the literature appears not to address the question directly (as implied in the review by Goldman and Dacre [1989]), it would seem that L would penetrate the skin much more readily than LO, doubtless hydrolyzing by the time it reached the circulatory system. As to comparisons of the abilities of these two substances to penetrate skin sufficiently to cause blistering, the statement has been made (HQ/DA,DN,AF 1990) that, "when humidity is high, lewisite hydrolyzes so rapidly that it is difficult to maintain a concentration sufficient to blister bare skin." The danger of absorption of LO through the skin may be negligible.

Inhalation of dust containing LO would be the most probable exposure route. The fact that the systemic toxicity of L is considerably less via the dermal route than by inhalation, intravenous injection, or subcutaneous injection suggests that a large portion of L gets trapped in the upper layers of the skin as LO and may act locally but does not reach the circulatory system. There is no evidence that LO, or any other organic arsenical, is carcinogenic, mutagenic, or teratogenic (Goldman and Dacre 1989).

As with L, the toxicity of LO may be attributed to an increase in capillary permeability to plasma proteins. That effect, in turn, appears to be due to the ability of the compound to interfere with such enzymes as pyruvate oxidase by binding with enzyme thiol groups (Goldman and Dacre 1989).

The only available datum on acute toxicity for LO is a subcutaneous LD_{lo} of 5 mg/kg in the mouse (Sweet 1987). This might be compared to a rat oral LD_{lo} of 50 mg/kg for 2-chlorovinylarsonic acid (Sweet 1987).

D. Discussion

LO is not regulated specifically by EPA under the RCRA program, although arsenicals (i.e., arsenic-containing chemical agents and associated compounds), including LO, are incorporated into 40 CFR, Part 261, Appendix VIII, under the general notation, "Arsenic Compounds, N.O.S. (not otherwise specified.)" LO is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)], nor is LO content treated as a basis for listing hazardous waste as F999 [R315-50-9(1)]. The Army suggested that LO be considered for this regulation since it is a breakdown product of L. Hence, it is evaluated herein.

The main differences between LO and L lie in their physicochemical properties. Depending on the methods used, chemical analyses of these compounds may fail to distinguish one from the other. Because LO is a breakdown product of L, it should not be considered as an acutely toxic commercial chemical product. Considering that LO is expected to be somewhat toxic, it should be considered as a hazardous constituent, however.

In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261, Appendix VIII hazardous constituent.

- Add as a 40 CFR 261, Appendix VII, basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Note also that arsenic-containing wastes not specifically listed as hazardous wastes may be classified as hazardous waste under the EPA toxicity characteristic (incorporated under DSHW regulation R315-2-9) as D004 if it leaches arsenic at levels greater than or equal to 5 mg/L.

LO is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14 because arsenic is expected to serve better in this capacity. Note that arsenic is already included in 40 CFR 264, Appendix IX, which is adopted in R315-50-14. Because arsenic is ubiquitous in the environment, and often present from anthropogenic activities, any monitoring program would be expected to detect a statistically significant increase over background concentrations. Under aqueous conditions, analyses should focus on CVAA and not LO.

24. Vx [S-2(2-Diethylamino)ethyl O-Isobutyl Methylphosphonothioate] CAS Reg. No. 159939-87-4

CH_3-P(=O)(OCH2CH[CH3]$_2$)(S-CH_2-CH_2-N[CH_2CH_3]$_2$)

A. Background

Vx is reputed to be the former Soviet Union's counterpart to VX. It is similar to VX chemically. Although not present in the U.S. stockpile or in any CAIS, some testing of this agent may be conducted.

B. Physicochemical Properties

The empirical formula for Vx is $C_{11}H_{26}NO_2PS$; its molecular weight is 267.37. Additional information on environmentally relevant properties of Vx is not available. Its behavior is expected to be similar to that of VX.

C. Toxicity

Like VX, Vx is a cholinesterase inhibitor (a "nerve agent"), with all the effects attributable to VX.

D. Discussion

Vx is not currently regulated by the EPA under the RCRA program. Furthermore, Vx is not currently regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. Vx content is not currently considered a basis for listing hazardous waste F999 [R315-50-9(1)]. The Army suggested that the status of Vx be considered, inasmuch as some testing with this chemical may be conducted. Hence, it is evaluated herein.

Because Vx is not in the U.S. stockpile or in any CAIS, it should not be considered as an acutely toxic commercial chemical product. It should be considered as a hazardous constituent, however. In accordance with the criteria defined in Section VII.A of the Preamble, the DSHW proposes the following:

- Add to R315-50-10 as a 40 CFR 261, Appendix VIII, hazardous constituent.

- Add as a 40 CFR 261, Appendix VII, basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

Since Vx would be expected to be used, if at all, only in very small quantities, it is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

25. CK (Cyanogen Chloride) CAS Reg. No. 506-77-4

Cl-CN

A. Background

CK is both a chemical agent and an industrial compound. As a toxic agent, CK was of interest mainly because at high concentrations it was able to challenge and degrade respiratory protection by charcoal filters, decreasing the ability to protect against other agents. CK was first used by the French in October 1916, but it was not employed extensively in World War I. It was adopted as a standard U.S. agent in World War II, but was not disseminated in combat. CK is not currently stockpiled by the Defense Department, but it is present in some CAIS.

B. Physicochemical Properties

CK is extremely volatile, disperses quickly unless confined, and tends to polymerize in the presence of certain impurities (Bodek et al. 1988). In water it is slowly hydrolyzed to cyanate (Bodek et al. 1988), a species of very low toxicity (Sweet 1987). At pH 4-6 and 11°C, the half-life would be about 3 months (based on Gmelin 1976); this reaction is accelerated by basic or strongly acidic conditions (Gmelin 1976). Table 31 presents environmentally relevant properties of CK. The following vapor-pressure-temperature equation is applicable to CK (Whitacre 1983):

$$\log P\ (\text{torr}) = 8.6642 - 1{,}654.6/T(K).$$

C. Toxicity

Table 32 shows some acute toxicities of CK in different mammalian species by two routes of administration. CK is highly irritating to eyes and mucous membranes. The general action of CK, interference with use of oxygen by the

TABLE 31 Environmentally Relevant Properties of CK

Property	Data	Reference
Empirical formula	CNCl	Not applicable
Molecular weight (MW), g/mol	61.48	Not applicable
Density, g/mL	1.20/10°C 1.18/20°C	Edgewood Arsenal 1974 HQ/DA,DN,AF, 1990
Melting point, °C	-6.9	HQ/DA, DN, AF 1990
Boiling point, °C	12.9	Edgewood Arsenal 1974
Vapor pressure at 25°C, torr	1,000	Edgewood Arsenal 1974
Log K_{ow} (estimate)	-1.21	
Aqueous solubility[a], g/L	69/20°C	Edgewood Arsenal 1974
K_H, atm·m^3/mol	9×10^{-4}/20°C	Estimated from the equation for log P (see text).

[a] Presumably at atmospheric pressure.

TABLE 32 Comparison of Some Acute Toxicities of CK by Different Routes of Administration in Mammalian Species

Route	Type of Exposure	Species	Toxicity
Oral	LD_{50}	Cat	6,000 µg/kg
Inhalation (mg/m^3/1 min)	LD_{50}	Cat	6,000 mg/m^3/1 min
Inhalation (mg/m^3/1 min)	LD_{50}	Dog	3,800 mg/m^3/1 min
Inhalation (mg/m^3/1 min)	LD_{50}	Monkey	4,400 mg/m^3/1 min

Source: Sweet (1987).

body tissues, is similar to that of hydrogen cyanide. However, CK differs from hydrogen cyanide in that it has strong irritating and choking effects and slows breathing (HQ/DA,DN,AF 1990). CK may cause pulmonary edema as a result of irritant action on the lungs. The human LCt_{50} is 11,000 mg·min/m^3 (HQ/DA,DN,AF 1990).

D. Discussion

CK is already regulated under the EPA P033 hazardous waste code, which is adopted in the State of Utah under R315-2-11(e). It is already considered an acutely toxic commercial chemical product that, on disposal, becomes a hazardous waste. CK is also listed in 40 CFR 261, Appendix VIII, which is adopted in R315-50-10. CK is not listed in 40 CFR 261, Appendix VII, as adopted in R315-50-9, as a basis for listing any F or K wastes. Finally, CK is not included in 40 CFR 264, Appendix IX, (Groundwater Monitoring Constituents), as adopted in R315-50-14. EPA included CK in its regulations, as indicated above, not because it is a chemical agent, but because it is a common industrial chemical. The Army suggested that the status of CK be considered because CK is present in some CAIS. Hence, it is evaluated herein.

CK is a fairly acutely toxic and irritating, but nonpersistent, chemical. On chemical transformation, such as hydrolysis, it largely loses its toxic properties. Within the domain of the U.S. government, other than in laboratory specimens, CK is found in some CAIS but not in stockpiled munitions.

In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Add as a 40 CFR 261, Appendix VII, basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

CK is also proposed to be added to R315-2-11(e) as an acutely toxic commercial chemical product P033, and to R315-50-10 as a hazardous constituent. Although already listed in EPA's regulations as such, which are adopted by the state, DSHW is proposing to list this chemical in R315-2-11(e) as P033 and in R315-50-10 for reasons of continuity and consistency.

Because CK rapidly degrades in water, it is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

26. CG (Phosgene or Carbonyl Chloride)
CAS Reg. No. 75-44-5

$C(=O)Cl_2$

A. Background

CG, a "choking" agent, as well as an important commercial industrial chemical, was first used in combat by the Germans against the British in Flanders during December 1915 and eventually was used by all the participants of World War I. It has not since been used in combat involving the United States. CG is extremely volatile and would not persist in the environment.

B. Physicochemical Properties

CG hydrolyzes rapidly in water to the relatively innocuous products carbon dioxide and hydrogen chloride, with a first order rate constant of about 6 s^{-1} at 25°C, and thus a half-life of about 0.12 second (Gmelin 1976). It also reacts readily with bases in aqueous solution (Clark 1989).

Environmentally relevant information is presented in Table 33. In addition to those data, the following vapor pressure-temperature (K) relationship has been reported (Whitacre 1983):

$$\log P\ (\text{torr}) = 7.460 - 1{,}289.2/T\ (\text{K}).$$

It may be concluded that once released to the environment and exposed to moisture, CG would rapidly hydrolyze to relatively harmless products.

C. Toxicity

Table 34 shows some inhalation LC_{50} values for different rodent species for a 30-minute period. CG is a lung irritant with cumulative effects. The human LCt_{50} is reportedly 3,200 mg·min/m^3 — for example, 100 mg/m^3 for 32 minutes. The human ICt_{50} is 1,600 mg·min/m^3. CG casualties are often delayed for several hours; there is little immediate reaction or irritancy. Exposure to extremely high concentrations may cause death in 5 hours or less (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990; EPA 1993).

Depending on exposure levels, initial symptoms of acute CG poisoning may be mild conjunctivitis, coughing, tightness in the chest, nausea and vomiting, and

TABLE 33 Environmentally Relevant Properties of CG

Property	Data	Reference
Empirical formula	CCl_2O	Not applicable
Molecular weight (MW), g/mol	98.92	Not applicable
Density, g/mL	1.373/20°C	HQ/DA,DN,AF, 1990
Melting point, °C	-127.8	Beilstein Sys. No. 199
Boiling point, °C	7.56	Beilstein Sys. No. 199
Vapor pressure at 25°C, torr	1,361	Whitacre 1983
Log K_{ow} (estimate)	-1.30	Estimated from the equation for log P (see text).
Aqueous solubility[a], g/L	6.8/one atm.	Clark 1989

headache. After a delay of 30 minutes to 24 hours, during which time symptoms have largely disappeared, pulmonary edema (filling of the lungs with fluid) may occur rapidly, along with burning of the throat and chest, fast shallow breathing, painful cough, spitting up of frothy blood-tinged sputum, and marked cyanosis (turning blue). Damage is to the capillaries, bronchioles, and alveoli of the lungs. Death is from shock or "drowning." Casualties from acute exposures who survive more than 48 hours usually recover without sequelae except for residual pulmonary deficit; however, bronchopneumonia and lung abscesses may develop (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990; EPA 1993).

TABLE 34 Acute Inhalation Toxicities of CG in Four Rodent Species

Species	30-Minute LC_{50}(mg/m^3)
Guinea pig	1,300
Mouse	1,300
Rat	1,300
Rabbit	1,300

Source: Sweet (1987).

Workers repeatedly exposed to low concentrations of CG exhibited disturbances in lung function, coughing, shortness of breath on exertion, and pain or tightness of the chest. Residual pulmonary deficit might be expected from chronic exposures (Edgewood Arsenal 1974).

D. Discussion

CG is already regulated under the EPA P095 hazardous waste code, which is adopted in the State of Utah under R315-2-11(e). It is already considered an acutely toxic commercial chemical product that, on disposal, becomes a hazardous waste. CG is also listed in 40 CFR 261, Appendix VIII, which is adopted in R315-50-10. CG is not listed in 40 CFR 261, Appendix VII, as adopted in R315-50-9, as a basis for listing any F or K wastes. Finally, CG is not included in 40 CFR 264, Appendix IX (Groundwater Monitoring Constituents), as adopted in R315-50-14. EPA included CG in its regulations, as indicated above, not because it is a chemical agent, but because it is a common industrial chemical. The Army suggested that the status of CG be considered because it is present in some CAIS. Hence, it is evaluated herein.

CG is an acutely toxic chemical. On chemical transformation, such as decontamination or environmental degradation, it loses its toxic properties. CG is no longer found in Defense Department stockpiles, though it is present in some CAIS.

In accordance with the criteria defined in Section VII.A, DSHW proposes the following:

- Add as a 40 CFR 261, Appendix VII, basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

CG is also proposed to be added to R315-2-11(e) as acutely toxic commercial chemical product P095, and to R315-50-10 as a hazardous constituent. Although already listed in EPA's regulations as such, which are adopted by the state, DSHW is proposing to list this chemical in R315-2-11(e) as P033 and in R315-50-10 for reasons of continuity and consistency.

Because CG rapidly degrades in water, it is not proposed for inclusion as a 40 CFR 264, Appendix IX, groundwater monitoring constituent in R315-50-14.

27. BZ (3-Quinuclidinyl Benzilate) CAS Reg. No. 6581-06-2 (HCl salt 13004-56-3)

$C_{21}H_{23}NO_3$

A. Background

The glycolate incapacitating agent BZ is a central nervous system depressant (as well as a hallucinatory drug) chosen as an agent for its ability to incapacitate rather than to kill. The ratio of the lethal level to the incapacitating level is high. BZ was introduced in the 1950s. BZ is considered a U.S. Department of Transportation (DOT) Class B poison (Edgewood Arsenal 1974). BZ is a relatively nonhazardous drug that is not part of the U.S. military chemical munitions stockpile or in any CAIS.

B. Physicochemical Properties

Table 35 lists several environmentally important properties of BZ. BZ contains three functional groups — hydroxyl, carboxylic ester, and tertiary amine. The first aids in its water solubility; the second is vulnerable to hydrolysis; the third aids in its water solubility and also confers basicity on it. The aqueous pK_a of BZH^+ at 25°C has been difficult to determine, with reasonable values ranging from 8.67 to 9.34; the most probable value (Rosenblatt et al. 1977) is about 9.0. The temperature (K)-dependent equation has been given as:

$$\log K_a = (-2455.8/T) - 1.0647.$$

TABLE 35 Environmentally Relevant Properties of BZ

Property	Data	Reference
Empirical formula	$C_{21}H_{23}NO_3$	Not applicable
Molecular weight (MW), g/mol	337.41	Not applicable
Density, g/mL	1.33	Safety Office undated
Melting point, °C	167.5	Safety Office undated
Boiling point, °C	412	Safety Office undated
Vapor pressure at 25°C, torr	1.43×10^{-7}	Rosenblatt et al. 1977
Log K_{ow}	2.32; 1.98	Estimates
Aqueous solubility[a], g/L	0.0118/25°C	Edgewood Arsenal 1974
K_H, atm·m^3/mol	5.4×10^{-12}/25°C	Estimated from the equation for log P (see text).
Log K_{oc}	2.24; 1.96	Estimated from the equation for log P (see text).

Hull et al. (1979) determined rate constant equations for four hydrolysis reactions that dominate in different pH ranges:

Protonated BZ with H_3O^+	$\log k_1$	$= (-3388.2/T) + 8.2477$
Protonated BZ with H_2O	$\log k_2$	$= (-2717.5/T) + 2.5827$
Protonated BZ with OH^-	$\log k_3$	$= (-2379.6/T) + 11.5558$
Unprotonated BZ with OH^-	$\log k_4$	$= (-2531.6/T) + 10.6599$

Vapor pressure equations are given by Rosenblatt et al. (1977):

For solid BZ, $\log P\ (\text{torr}) = 17.2577 - (8080.4/T[K])$.

For liquid BZ, $\log P\ (\text{torr}) = 9.6412 - (4631.7/T[K])$.

C. Toxicity

Table 36 shows LD_{50} values in two mammalian species. The term "incapacitating agent" applies to a substance for which the ratio between the directly lethal level and the threshold incapacitation level is relatively high. This criterion is reasonably applied to BZ because the estimated human LD_1 (dose lethal to 1% of an exposed population) is in the range of 200-1,400 µg/kg (Rosenblatt et al. 1977), and the onset of symptoms occurs at a dose of about 1-5 µg/kg (Byrd et al. 1987). The inhalation ICt_{50} for a BZ aerosol (HQ/DA,DN,AF 1990) (with particles of 0.8 µm mass median diameter) is 112 mg·min/m^3. Daily doses of

TABLE 36 Comparison of Some Acute Toxicities of BZ by Different Routes of Administration in Two Species

Route	Species	LD_{50} (Base) (mg/kg)	LD_{50} (Hydrochloride) (mg/kg)
Intravenous	Mouse	25	18
Intravenous	Dog	-[a]	15
Intravenous	Mouse	-	110

[a] "-" = not determined.

Source: Sweet (1987).

1 µg/kg produce no evidence of cumulative effects (Rosenblatt et al. 1977). The mental impairment that is an aspect of BZ incapacitation (as with similar drugs) could lead to serious and possibly fatal consequences for an affected individual. The effects of BZ on mental performance were shown to persist for at least 2-3 weeks in humans (Rosenblatt et al. 1977). Routes of entry into the body are primarily by inhalation and ingestion; BZ can be absorbed dermally with the proper solvent, and there may be local ocular effects (Safety Office undated).

BZ is an anticholinergic agent (like atropine). Small doses cause sleepiness and reduced alertness, decreased ability to remember, to solve problems, to pay attention or to understand instructions, increased heart rate, dry and flushed skin and lips, mydriasis (enlarged pupils), urinary retention, constipation, and elevated skin temperature. Larger doses produce additional signs of intoxication: increased blood pressure, extreme excitement, dizziness, involuntary muscular movements, vomiting, blurred vision, confusion, delusions and delirium, and finally sedation leading to stupor and inability to respond effectively to the environment. During the recovery phase, there is increasing activity and random unpredictable behavior, with a gradual return to normal in about 12-96 hours (Edgewood Arsenal 1974; HQ/DA,DN,AF 1990; Rosenblatt et al. 1977; Safety Office undated).

A review of the histories of human volunteers exposed to anticholinergic agents (Panel on Anticholinesterase Chemicals and Panel on Anticholinergic Chemicals 1982), including BZ, concluded that, "No firm evidence has been seen that any of the... test compounds surveyed produced long-range adverse human health effects at the doses used at Edgewood Arsenal."

D. Discussion

BZ is not currently regulated under the RCRA program by EPA and is not currently incorporated into DSHW regulations. Thus, BZ is not regulated under the State of Utah P999 hazardous waste code [R315-2-11(e)]. Neither is BZ content recorded as a basis for listing hazardous waste as F999 [R315-50-9(1)]. DSHW suggested including BZ in a draft rule it released to the Army for review in February 1996. Hence, it is evaluated herein.

BZ, as an incapacitating agent, is moderately toxic. BZ is not in Defense Department stockpiles or in any CAIS, although there may yet be a few surviving non-stockpile rounds containing it, and it may be used in small amounts for research purposes. It is available in small amounts, primarily at research facilities, such as at Dugway Proving Ground. BZ may, therefore, be considered a toxic commercial chemical product. In accordance with the criteria defined in Section VII.A of the Preamble, DSHW proposes the following:

- Define as a 40 CFR 261.33(e) commercial chemical product U902 in R315-2-11(e).
- Add to R315-50-10 as a 40 CFR 261 Appendix VIII hazardous constituent.
- Add as a 40 CFR 261 Appendix VII basis for listing specific newly proposed hazardous wastes K901 through K908 in R315-50-9(1).

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